ARCHIVES OF OTOLARYNGOLOGY

VOLUME 30 AUGUST 1939 NUMBER 2

COPYRIGHT, 1939, BY THE AMERICAN MEDICAL ASSOCIATION

NEUROLOGIC COMPLICATIONS OF INFECTIONS OF TEMPORAL BONE AND PARANASAL SINUSES

SUMMARY OF TWENTY YEARS' (1919 TO 1938) EXPERIENCE

JOSEPH C. YASKIN, M.D.
PHILADELPHIA

OUTLINE

Introduction
Material
Anatomic Considerations and Routes of Infection
General Clinical Considerations
Conditions with Predominantly Meningeal Reactions
    Pachymeningitis Externa and Extradural Abscess
    Pachymeningitis Interna and Subdural Abscess
    Circumscribed Leptomeningitis
    Protective Meningitis
    Bacterial Leptomeningitis
Abscess of the Brain
    Pathology and Pathogenesis
    Clinical Types
    Abscess of the Temporal Lobe
    Abscess of the Frontal Lobe
    Other Cerebral Abscesses
    Cerebellar Abscess
    Treatment of Abscess of the Brain
Nonsuppurative Encephalitis
Otitic Hydrocephalus and Arachnoiditis
Thrombosis or Phlebitis of the Intracranial Venous Channels
    Thrombosis of the Lateral Sinus
    Thrombosis of the Cavernous Sinus
    Thrombosis of the Superior Longitudinal Sinus
Cranial Nerves
    Optic Nerve
    Nerves to the Extraocular Muscles
    Oculomotor Nerve
    Abducens Nerve
    Trigeminal Nerve
    Facial Nerve

From the Department of Neurology, Graduate School of Medicine, University of Pennsylvania.

Read in part as the presidential address before the Philadelphia Neurological Society, Jan. 27, 1939.

Summary and Conclusions
Appendix: Report of Nineteen Cases

INTRODUCTION

With few organic conditions of the nerves is prompt and accurate diagnosis of greater importance than with certain neurologic complications arising in diseases of the temporal bone and the accessory nasal sinuses. These complications tax the ingenuity of the otorhinologist, who is anxious to obtain every assistance from the neurologist and the ophthalmologist in order to determine the course of treatment. With few neurologic conditions is an accurate diagnosis more difficult to make than with these complications. The difficulties in diagnosis are traceable to a number of factors:

1. The complexity of the anatomic structures and relations in and about the temporal bone and paranasal sinuses.

2. The variations in the anatomic structures, especially in the extent of pneumatization and of dehiscences.

3. The complexity of the routes of infection by which the endocranial contents and the various peripheral nerves are implicated.

4. The paucity and variability of clinical manifestations in a fair percentage of cases and the absence or masking of signs in some cases.

These and other factors are familiar to the neurologist who has had any experience with otorhinologic practice. They become more vividly familiar in the course of years, as errors in observation and judgment accumulate. The otorhinologist is frequently aware of the shortcomings of the clinical neurologist. This was stressed by Langenbeck[1] and by Ramadier, Caussé, André-Thomas, Barré and Velter.[2] Langenbeck pointed out that the otologist has a different attitude toward intracranial disease processes than does the neurologist. Whereas the otologist usually sees rapidly developing disorders, the neurologist more frequently observes slowly developing tumor of the brain. He further stated that in cases of comparatively early, that is, not yet well defined, inflammation in the region adjoining the cranial contents, the neurologist may discover numerous symptoms which are of no importance in relation to the disease process as a whole. Especially in cases of acute exacerbation, the results of neurologic examination may suggest the presence of an endocranial focus, but the otologist need not become alarmed, since the course of the disease and the patient's general condition are satisfactory. On the other hand, in cases of a severe endocranial focus of inflammation, the neurologic examination may supply no information regarding the localization. Langenbeck concluded that

1. Langenbeck, B.: Otiatrisch-neurologische Erfahrungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:63, 1935.

2. Ramadier, J.; Caussé, R.; André-Thomas; Barré, J. A., and Velter, E.: Les abcès du cervelet, Rev. d'oto-neuro-opht. **13**:1 and 85, 1935.

if the systemic manifestations are marked and the entire clinical aspect suggests an endocranial complication the otologist should make use of the suggestions of the neurologist regarding localization. All are familiar with the predicament of the clinician when confronted with the observations reported by the pathologist. Influenced by such experience and by the findings in the study presented here, I am of the opinion that the neurologist should be consulted by the otorhinologist as soon as neurologic complications are suspected.

These considerations prompted the review of the experience of twenty years (1919 to 1938) with the neurologic complications of otitis and diseases of the paranasal sinuses, with the thought that the data obtained in such a study may be useful for the neurologist, the otorhinologist and the ophthalmologist.

TABLE 1.—*Summary of Material (1919-1938)*

Condition	Origin of Neurologic Complications, Number of Cases			Total
	Temporal Bone	Paranasal Sinus	Other Sites	
Extradural abscess	9	5	..	14
Subdural abscess	3	4	..	7
Protective meningitis	9	2	..	11
Bacterial meningitis	56	31	109	196
Abscess of brain	30	16	..	46
Nonsuppurative encephalitis	2	3	..	5
Otitic hydrocephalus	2	..	..	2
Adhesive arachnoiditis	..	..	1	1
Thrombosis of lateral sinus	19	..	..	19
Thrombosis of cavernous sinus	2	2	7	11
Thrombosis of superior longitudinal sinus	2	2	..	4
Papilledema	26	13	..	39
Oculomotor palsy	5	7	..	12
Palsy of abducens nerve	13	6	..	19
Gradenigo syndrome	5	..	..	5
Petrositis	8	..	..	8
Peripheral paralysis of facial nerve	42	..	..	42
Hunt syndrome	2	..	..	2
Total	235	91	117	443

MATERIAL

The material for the presentation was obtained from the Graduate Hospital, from Northern Liberties Hospital and from private practice. A large majority of the cases was observed by me. Of the cases not observed by me, only those with satisfactory clinical or pathologic records were included in this study. The diagnosis in the majority of cases was confirmed by operation, by reliable clinical tests or by autopsy.

The material is summarized in table 1. In discussing the material I shall stress only factors which have a bearing on the diagnosis and treatment, and the literature will be reviewed only for purposes of clarifying these factors.

Prior to analyzing my material I deem it advisable, in the interest of conciseness and for better clinical orientation, to make some general remarks concerning certain established and useful data on the anatomy of the temporal bone and paranasal sinuses and the routes of infection in, and the diagnosis and treatment of, the majority of their diseases. Specific factors will be discussed in the analysis of individual conditions.

Nineteen cases were deemed worthy of reporting and are abstracted at the end of the article. Some of these were chosen from an array of cases (326) for illustrative purposes and others because of their rarity or of some special interest. The principal features of these cases are summarized in table 2.

ANATOMIC CONSIDERATIONS AND ROUTES OF INFECTION

The intracranial contents may be invaded: (*a*) by continuity and contiguity, through bony defects consequent to osteomyelitis, local

TABLE 2.—*Summary of the Nineteen Cases Abstracted in This Series*

Case	Initals	Diagnosis	Comment
1	W. T.	Abscess of frontal lobe of brain	No sinal disease on admission to hospital
2	A. S.	Meningitis	Causeless vomiting and mastoiditis without aural discharge
3	K. G.	Subarachnoid hemorrhage	Associated with sinusitis and diagnosed as meningitis
4	C. H.	Subdural hematoma	Sinal disease and history of trauma not elicited until after recovery
5	R. B.	Tumor of brain	Optic neuritis ascribed to associated severe sinal disease
6	W. B.	Subdural abscess and sinus thrombosis	Antecedent labyrinthine symptoms pointing to localization in posterior fossa
7	P. S.	Multiple renal abscesses	Undiagnosed marked delirium following severe sinal infection
8	J. T.	Extradural abscess	Increased intracranial pressure relieved by mastoidectomy
9	B. S.	Protective meningitis	Pachymeningitis and protective meningitis; recovery with sulfanilamide
10	M. B.	Abscess of frontal lobe of brain	Abscess contralateral to sinal disease
11	B. G.	Otitic nonsuppurative encephalitis	Abscess of temporal lobe of brain diagnosed and explored for; recovery after mastoidectomy
12	E. K.	Otitic nonsuppurative encephalitis	Aphasia; recovery after mastoidectomy
13	A. B.	Localized nonsuppurative meningoencephalitis	Condition contralateral to sinal disease
14	L. B.	Generalized nonsuppurative encephalitis	Condition following mild sinusitis
15	W. L.	Nonsuppurative encephalitis	Involvement of multiple cranial nerves; alkalosis
16	H. B.	Adhesive arachnoiditis simulating tumor of brain	Long duration with periods of remission
17	E. L.	Otitic hydrocephalus	Condition following ligation of jugular vein
18	A. McC.	Otitic hydrocephalus	Recovery following reoperation and removal of necrotic bone
19	A. K.	Subdural abscess on right with thrombosis of left cavernous sinus	Unsuspected suppuration of left petrosa

erosion of bone or fracture; (*b*) through congenital defects in the adjacent bony walls; (*c*) by extension through preformed pathways; (*d*) by way of blood vessels, especially veins, or (*e*) by way of perineural sheaths. The lymphatic system plays no role, Turner and Reynolds.[3]

To understand the pathways of infection it is indispensable to have an intimate knowledge of the anatomy of the temporal bone and the paranasal sinuses. For this the reader is referred to standard books on anatomy. However, by studying the pathways of infection a better

3. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Diseases: A Pathological and Clinical Study of the Pathways of Infection from the Face, the Nasal and Paranasal Air Cavities, Edinburgh, Oliver & Boyd, 1931.

appreciation is gained of the intricate anatomic structures and relations; i. e., the mode of the extension of the infection cannot be understood without anatomic orientation.

Schillinger [4] stated that the intracranial contents may be invaded from the temporal bone in the following ways:

A. Through the middle ear and antrum
   1. Upward, through the tegmen, either along the system of venules which traverse the tegmen to communicate with the veins of the dura or by contiguity and through dehiscenses in the bone; spread of infection in this direction eventuates in an epidural abscess, an abscess of the temporosphenoid lobe or meningitis
   2. Inward or medially
      a. Through the labyrinth the spread is along the sheaths of the seventh and eighth nerves, through the internal auditory meatus to the meninges of the posterior fossa or else through the aqueduct of the cochlea or the aqueduct of the vestibule
      b. Around the labyrinth, into the petrous pyramid, thence through the apex to the meninges
   3. Downward, by venous channels running from the middle ear into the jugular bulb, whence, by means of retrograde phlebitis or of thrombosis, the spread of infection reaches the meninges

B. Through the mastoid cells
   1. Upward to the middle fossa (as described in A 1)
   2. Inward, by way of the perilabyrinthine cells to the petrous pyramid and thence through the apex to the meninges
   3. Posteriorly, through the inner table to the cerebellum, producing cerebellar abscess or meningitis of the posterior fossa
   4. In various directions to reach the sigmoid sinus, producing perisinuous abscess, sinus phlebitis or sinus thrombosis
   5. Downward to the jugular bulb by spread through the peribulbar cells

The modes of invasion into the intracranial contents from diseases of the paranasal sinuses are even more complicated. The rich venous connections of the frontal, ethmoid, sphenoid and maxillary sinuses facilitate extension by thrombophlebitis not only to adjacent structures in the anterior and the middle cranial fossa but also to the cavernous and even the superior longitudinal sinus (Courville and Rosenvold [5]). Extension along the perineural spaces about the olfactory nerves is probable. Osteomyelitis or osteitis of the frontal, ethmoid, sphenoid or maxillary sinus frequently causes extension into adjacent structures. An important anatomic factor in intracranial complication is the structure of the temporal bone and the sphenoid sinuses. The work of

4. Schillinger, R.: Otitic Meningitis, Arch. Otolaryng. **25**:455 (April) 1937.

5. Courville, C. B., and Rosenvold, L. K.: Intracranial Complications of Infections of Nasal Cavities and Accessory Sinuses: Survey of Lesions Observed in a Series of Fifteen Thousand Autopsies, Arch. Otolaryng. **27**:692 (June) 1938.

Onodi,[6] Sluder,[7] Schaeffer,[8] Wittmaack[9] and others has demonstrated that the degree of pneumatization, the size and symmetry of the air cavities and the thickness of the walls determine the facility of the extension of infection. Arrangements of vascular channels, the presence of certain canals, such as the craniopharyngeal, and dehiscenses contribute to the spreading of infection.

GENERAL CLINICAL CONSIDERATIONS

The otorhinologist often confronts the neurologist with some of the following questions: Is there a neurologic complication? Is it extradural, subdural, in the leptomeninges, in the brain or in the venous sinuses, or does it involve the cranial nerves only? What studies are indicated? Is it necessary, or safe, to do a lumbar puncture? Are air studies indicated, and if so what kind? Should surgical intervention be instituted, and if so when and by what route?

To answer these and other questions, the neurologist must have a thorough familiarity with the anatomic, pathologic and clinical factors of the problem, and he should also bear in mind that in addition to making complete neurologic examinations he must take into account not only the customary neurologic symptoms but also all historical data which can be procured. The important factors follow:

(*a*) The duration, mode of onset and chronologic development of the various symptoms. Chronic infections are more likely to give rise to intracranial complications, especially abscess of the brain, than are acute processes, although an acute exacerbation may be the determining cause of the complications. It is worthy of note that the original infection of the paranasal sinuses may heal, leaving no objective signs of previous infection, yet intracranial complications may develop, as is seen in case 1. However, the nasopharyngeal symptoms may be mild and transient and yet be followed by gross cerebral complications. Intracranial infections may follow lesions of the temporal bone without perforation of the drum and without mastoiditis.[10] Acute mastoiditis may develop in old otitis without aural discharge, as in case 2.

6. Onodi, L.: The Rhinogenic and Otogenic Lesions of the Third, Fourth, Fifth and Sixth Cranial Nerves, J. Laryng., Rhin. & Otol. **14**:304 and 360, 1914.

7. Sluder, G.: Concerning Some Headaches and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Company, 1918.

8. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasocranial Passages and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

9. Wittmaack, K.: Ueber die Pneumatization des Schlafenbeines, Jena, Gustav Fischer, 1918.

10. Blum, I.: Ein Fall von Hirnabscess otogenen Ursprunges bei unperforierten Trommelfell, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **19**:638, 1932. Love, A. A.: Complications of Otitis Media Without Rupture of the Membrana Tympani, Arch. Otolaryng. **17**:297 (March) 1933.

The neurologist should remember also that the history or existence of nasoaural infection need not indicate that coexistent endocranial lesions are otorhinogenic, as is exemplified in cases 3, 4 and 5, in which the lesions were associated respectively with spontaneous subarachnoid hemorrhage, chronic subdural hematoma and tumor of the brain.

The development of *new* symptoms may help in determining the localization of the lesion. Thus, a history of a sudden development of vertigo with nausea and vomiting tends to indicate that labyrinthitis has antedated endocranial disturbances and that the neurologic complications are more likely to occur in the posterior fossa, as exemplified in case 6.

(*b*) The findings, previous operations and present local conditions should routinely be inquired into. Thus, the finding at a previous operation of local pachymeningitis, epidural collections, fistula or involvement of a venous sinus may help in evaluating the existing findings.

(*c*) Careful inquiry and observations should be made with reference to pulse, blood pressure, temperature, respiration, blood counts and other data on the systemic condition of the patient. However, neuropsychiatric symptoms may be due not to direct involvement of the brain but no visceral disease, as in case 7, in which persistent delirium was due to multiple renal abscesses.

(*d*) The clinician should remember that lesions in the temporal bone and paranasal sinuses may be multiple. Thus, the attention is often centered on one temporal bone while lesions in the opposite temporal bone or the sinuses are overlooked; yet they may be the cause of the neurologic complications. This is well illustrated in case 19, detailed in a later section. It cannot be too strongly stressed that sinal infection frequently accompanies disease of the temporal bone and may be independently the cause of the neurologic complications.

(*e*) I have come to the conclusion that whenever there is any doubt, the spinal fluid should be studied. I have seen no harm resulting from such a procedure and am furthermore convinced that diagnosis and treatment are frequently delayed by the failure to perform lumbar puncture in the early stage of an endocranial complication. The lumbar puncture should be performed cautiously, always with the patient lying down and always with a manometer, preferably a water manometer, for if the pressure is over 250 mm. of water only a small amount of fluid is to be withdrawn, for a cell count. If abscess of the brain is not suspected, the withdrawal of larger amounts of fluid is permissible and, in fact, indicated.

(*f*) The general principles underlying the treatment of neurologic complications include: (1) eradication of infectious foci and establish-

ment of satisfactory drainage (this holds true even with the success of the latest chemotherapy [11]); (2) attacking the intracranial complication directly, as in abscess of the brain, which will be discussed in a later section; (3) use of sulfanilamide and related drugs when possible, and (4) supportive and symptomatic measures.

The individual neurologic conditions will now be discussed.

CONDITIONS WITH PREDOMINANTLY MENINGITIC REACTIONS

Pachymeningitis externa and extradural abscess, pachymeningitis interna and subdural abscess, circumscribed leptomeningitis, protective meningitis, bacterial leptomeningitis, meningismus and arachnoiditis have predominantly meningitic reactions. The last-named condition will be discussed in a later section.

Pachymeningitis Externa and Extradural Abscess.—Inflammation of the outer surface of the dura and collection of pus between the dura and bone occur rather frequently, and no definite figures can be given regarding their incidence. These conditions probably occur more frequently in chronic than in acute disease of the temporal bone and paranasal sinuses. The avenues of infection are usually by continuity, from lesions of the temporal bone. Among lesions of the sinuses, Courville and Rosenvold [5] found these complications most commonly in association with frontal sinusitis. They offered the following explanation:

> The dura behind the posterior wall . . . presents a broad exposure to infection and is less adherent to the bone than at other points. Furthermore, its veins communicate with the veins in the bone and mucous membrane of the frontal sinus.

Perhaps the most common single factor is the presence of osteomyelitis of the frontal bone.

The only subjective symptom of dural change may be headache, which in my experience is usually more pronounced at night. Its location varies with the location of the lesion. When the tip of the petrous bone is involved, retro-ocular pain is the rule. When the lesion is over the tegmen the headache is temporal, and when in the posterior fossa the headache is in the occipital region. The headache may be absent.

Vertigo, nystagmus and other labyrinthine signs may be present in addition to headache when the lesion is in the posterior fossa, especially in association with thrombosis of the lateral sinus. Usually there are no localizing neurologic signs or evidences of increased intracranial pressure to permit a diagnosis of extradural abscess. Rarely epidural collections may cause symptoms resembling those of an abscess of the brain, as in case 8.

---

11. Schenck, H. P.: Use of Sulfanilamide in Otolaryngology: A Review of the Literature, Arch. Otolaryng. **28**:698 (Nov.) 1938.

In this series there were 14 cases of extradural abscess. It was impossible to evaluate the number of cases of uncomplicated pachymeningitis externa, which was frequently encountered during mastoidectomy.

Of the 14 cases, the lesion was associated with disease of the temporal bone in 9. Of these 9, 6 were fatal, the condition being associated with abscess of the temporal lobe in 1, with subdural abscess in 2 and with bacterial meningitis in 2. In the sixth fatal case endocranial complication did not occur. In 3 cases, the condition was uncomplicated, and the patient recovered.

Five cases in which the lesion was associated with disease of the frontal sinuses were all fatal. In 2 there was associated thrombosis of the superior longitudinal sinuses. In 3 of the cases some evidences of meningeal irritation appeared, and in 1 of them a large abscess of the frontal lobe developed, from which the patient recovered only to succumb a year later to generalized streptococcic meningitis.

The treatment of extradural abscess is entirely surgical. In fact, the very diagnosis of this condition can be made only by exploration.

Pachymeningitis Interna and Subdural Abscess.—Inflammation of the inner surface of the dura and collection of pus beneath the dura are even less frequent and more difficult to diagnose than pachymeningitis externa and extradural abscess. They occur usually with acute and subacute diseases of the temporal bone or the paranasal sinuses. Extension from pachymeningitis externa and extradural abscess, with or without dural fistula, is the usual mode of infection. The symptoms are essentially the same as in pachymeningitis externa and epidural abscess. It is probable that pachymeningitis interna forms the localized leptomeningitis responsible in some cases for protective meningitis, as is well illustrated by case 9.

In 7 cases of this series the diagnosis was subdural abscess. In 3 of these the lesion was associated with disease of the temporal bone, and in 2 there was associated fatal extradural abscess. In 4 cases the lesion was associated with frontal sinusitis. In these, 3 patients recovered and 1 died.

The treatment of subdural abscess is surgical. Exploration is the only way to an accurate diagnosis.

Circumscribed Leptomeningitis.—Kerrison [12] stated:

> This term is used to describe a suppurative inflammation involving the arachnoid and pia mater over a distinctly limited area, lateral extension being prevented by marginal adhesions binding the arachnoid and pia together and to the outer dural layer.

12. Kerrison, P. D.: Diseases of the Ear, ed. 4, Philadelphia, J. B. Lippincott Company, 1930, p. 385.

Circumscribed leptomeningitis is a condition which is probably responsible for most of the cases of protective meningitis.

Protective Meningitis.—Aseptic (sympathetic, protective or reactive) meningitis is probably the type of greatest importance to otorhinologists. Clinically, it is manifested by rigidity of the neck, the Kernig sign, some elevation in temperature, an increase in the pressure of the spinal fluid, which is clear or opalescent, an increase of cells (usually polymorphonuclears and rarely lymphocytes), an increase in total proteins, no appreciable change in the sugar or the chloride content and abscence of organisms. This condition is occasionally observed in the absence of any disease of the ear or the accessory sinuses. When disease is present, it may be associated with epidural abscess, circumscribed meningitis, intracerebral or cerebellar abscess or sinus thrombosis, or it may be the beginning of bacterial meningitis, before the bacteria are detectable by present laboratory procedures. I have observed an exceedingly large number of cases in which the condition was first diagnosed as aseptic meningitis, but my records reveal only 11 cases in which recovery followed without the finding of bacteria in the spinal fluid or the development of other intracranial complications. Of these 11 cases, the disease was associated with mastoiditis in 9, in 4 of which it preceded and in 5 of which it followed mastoidectomy, requiring revision of the operative field in 4. In the remaining 2 cases the condition was associated with infection of multiple sinuses, and recovery followed without any major surgical procedure. In the vast majority of cases in which a diagnosis of aseptic meningitis was made at the beginning, the condition was proved by subsequent studies of the spinal fluid to be bacterial meningitis, while in some there was either abscess of the brain, epidural abscess or sinus thrombosis.

The early diagnosis of aseptic meningitis is obviously desirable for therapeutic reasons. The absence of organisms is the chief differential point. However, a decrease in the sugar content and, according to Kopetzky and Fishberg,[13] "an increase in the potassium salts, a lower p_H, the lowered carbonate content . . . and, . . . particularly, the marked increase in the lactic acid content of the [spinal] fluid" favor the diagnosis of bacterial meningitis. Kopetzky[14] stated that these chemical changes may indicate invasion without actual tissue reaction (meningitis) and that after complete eradication of the foci of origin recovery may follow without the development of meningitis.

On the basis of experience in the past two years, there can be little doubt that the use of sulfanilamide and related products will reduce the

13. Kopetzky, S. J., and Fishberg, E.: Early Diagnosis in Meningitis: Chemical Studies of Spinal Fluid, Arch. Otolaryng. **16**:851 (Dec.) 1932.

14. Kopetzky, S. J.: The Clinical Picture of Bacterial Meningitis with Particular Reference to Its Changing Phases, Laryngoscope **47**:301, 1937.

frequency of development of bacterial meningitis if the existence of protective meningitis is recognized early in a given case. It should be stressed that the eradication of foci of infection is just as important now as it was before the days of newer chemotherapeutic agents.[11]

Bacterial Leptomeningitis.—In a previous publication (Yaskin [15]), which included consideration of cases from 1920 to 1937, mainly from the Graduate Hospital, bacterial leptomeningitis was considered in part. Table 3 includes the cases observed from 1919 to 1938. A glance at this table reveals at once that meningitis is otitic or rhinogenic in a considerable proportion of all cases. Statistics taken from large metropolitan districts (Williams and Gasling [16]) indicate that meningococcic and tuberculous types are most frequently observed.

In this series otitic meningitis is almost twice as frequent as rhinogenic meningitis. Yet it is of sufficient importance to note that meningitis

TABLE 3.—*Analysis of Cases of Bacterial Meningitis Observed from 1919 to 1938*

Organism	Origin				Total
	Systemic or Unknown	In Temporal Bone	In Paranasal Sinuses	From Cranial Trauma	
Meningococcus	19	..	..	..	19
Tubercle bacillus	39	..	..	..	39
Pneumococcus	13	21	9	4	47
Streptococcus	7	20	9	8	44
Influenza bacillus	4	..	..	..	4
Staphylococcus	4	4	6	3	17
Friedländer's bacillus	1	..	..	..	1
Unknown	4	11	7	3	25
Total	91	56	31	18	196

is the most common intracranial complication of disease of the paranasal sinuses.

Routes of Infection: (*a*) The routes of infection in otogenic meningitis are classified by Goerke [17] as follows:

A. Direct or Primary Meningitis (direct extension from the temporal bone)
1. Hematogenous form, by way of the pial veins
2. Tympanogenous form
   (*a*) By normal preformed paths
   (*b*) Through developmental preformed channels
   (*c*) Through pathologically developed paths

15. Yaskin, J. C.: Differential Diagnosis of Bacterial Meningitis of Aural and of Nasal Origin, Arch. Otolaryng. **27**:444 (April) 1938.

16. Williams, A. W., and Gasling, R.: Bacteriology of Meningitis, A. Research Nerv. & Ment. Dis., Proc. **12**:353, 1932.

17. Goerke, cited by Courville, C. B., and Nielsen, J. M.: Fatal Complications of Otitis Media, with Particular Reference to Intracranial Lesions in a Series of Ten Thousand Autopsies, Arch. Otolaryng. **19**:451 (April) 1934.

3. Labyrinthinogenous form
   (*a*) By preformed paths
   (*b*) By pathologically developed paths

B. Indirect or Secondary Meningitis (by way of other intradural complications)
1. Direct transmission from
   (*a*) Thrombosis of the lateral sinus
   (*b*) Subdural infection
   (*c*) Abscess of the brain
2. Concomitant or symptomatic meningitis associated with thrombosis of the lateral sinus, subdural infection or abscess of the brain

(*b*) The routes of infection in rhinogenic meningitis have already been discussed. Meningitis may be secondary to any septic lesion in the nose and sinuses and may constitute the only intracranial complication (primary in a sense). In some cases it may be coincident with or secondary to some other intracranial lesion, such as phlebitis of a venous sinus or abscess of the brain. Septic meningitis occurs most commonly secondary to sphenoiditis and ethmoiditis; less commonly, it follows inflammation of the maxillary or frontal sinus and develops only through the secondary lesions. It has followed submucous resection, the infection traveling by the olfactory perineural spaces.

Diagnosis: Bacterial meningitis is to be suspected in the presence of the *syndrome of meningeal irritation.* The common symptoms (headache, dizziness and vomiting) and focal signs of meningeal irritation (rigidity of the neck and Kernig's and Brudzinski's sign) are well known but require careful clinical evaluation. For instance, rigidity of the neck is often observed in normal infants. I have observed rigidity of the neck in 2 cases of early cervical adenitis, in which the diagnosis of meningitis was seriously considered. Recently a "Kernig sign" was found to be due to scurvy. In older persons arthritis of the spine may give rise to pseudomeningeal signs. On the other hand, I have observed 2 cases of purulent meningitis (streptococcic in both) and at least 3 cases of tuberculous meningitis in which at the height of the disease there was no rigidity of the neck and no Kernig or Brudzinski sign. In cases in which the diagnosis is doubtful, a lumbar puncture is indispensable for accurate diagnosis. It is generally known that meningeal signs may be absent early in the course of meningitis and that they often disappear in the terminal stages.

In the final analysis, the examination of the spinal fluid is of considerable aid in determining the cause of meningeal irritation. There are many basic causes for the various changes in the spinal fluid. Of these, three are most important: changes in the blood plasma, changes in the hematoencephalic barrier and the invasion of the ventriculosubarachnoid cavities by foreign elements. The condition is characterized by an elevation of the pressure of the spinal fluid and occasionally by an increase in the protein content, but the cell count remains normal, and organisms

are absent. Meningismus is seen in association with toxic conditions, such as delirium tremens, uremia and congestive cardiac disease. Meningismus is always observed when air is injected into the subarachnoid ventricular system.

The diagnostic significance of various findings in the spinal fluid was discussed in another presentation (Yaskin [15]). Kopetzky [18] stated that in early stages of meningitis there is always an increase in the lactic acid content of the spinal fluid, accompanied by a decrease of the alkali reserve, with a resultant lowering of the p_H from 7.5 to 6.9, and a reduction in the carbonates as a result of the loss of carbon dioxide from the increased lactic acid content.

Bacterial meningitis of otitic or nasal origin should be differentiated from (*a*) bacterial infections not of aural or nasal origin, especially meningococcic and tuberculous meningitis and from (*b*) nonbacterial meningitis, i. e., *acute meningeal symptoms due to agents other than known bacteria.* These include the following conditions:

1. Cervical adenitis and retropharyngeal abscess. The chief source of error in connection with these conditions is the limitation of movements of the neck. One rarely sees rigidity of the neck and the back in association with a tumor of the basal and the posterior fossa of the brain or with a spinal epidural abscess or tumor. Infiltration of the meninges by lymphoblastomas (Hodgkin's disease, leukemia) may rarely give rise to meningeal irritation.

2. Meningismus. Acute serous meningitis, or meningismus, occurs in the course of acute systemic infections, such as pneumonia, measles, influenza, gastroenteritis, acidosis, tetany and bacterial endocarditis.

3. Subarachnoid hemorrhage. In hemorrhagic conditions free blood in the subarachnoid space gives rise to acute meningeal symptoms. In 2 cases I have observed spontaneous subarachnoid hemorrhage in association with acute sinusitis which clinically closely resembled acute bacterial meningitis. Spontaneous subarachnoid hemorrhage is generally due to congenital aneurysm and in recent years has been common. Subarachnoid bleeding is observed most frequently in association with cranial trauma or with intracerebral hemorrhage and less commonly with purpura, hemophilia, drug or metal poisoning or severe systemic infection.

4. Virus diseases. Poliomyelitis, epidemic encephalitis, disseminated encephalomyelitis and benign lymphocytic choriomeningitis are associated with varying degrees of meningeal irritation. In cases of poliomyelitis there is in the first forty-eight hours a definite increase in cells in the spinal fluid. In cases of epidemic encephalitis and of disseminated encephalomyelitis there is usually no pleocytosis. Benign lymphocytic choriomeningitis occurs in epidemic form and is characterized by an

18. Kopetzky, S. J.: Notes on the Diagnosis of Otitic Meningitis, Ann. Otol., Rhin. & Laryng. **43**:401, 1934.

acute onset of meningeal signs, a marked increase of lymphocytes in the spinal fluid with absence of bacteria, a short, mild course without any complications and termination in from seven to ten days, usually in recovery, although some fatal cases have been reported.

5. Syphilis. Acute meningeal involvement in the course of syphilis is common but is usually patchy and chronic and is not accompanied by evidence of acute meningitis. Evidences of acute meningitis in cases of secondary syphilis are not uncommon, while the disease occurs in 5 per cent of cases of latent syphilis. In acute syphilitic meningitis rigidity of the neck, the Kernig sign, an increase of cells (predominantly lymphocytes), changes in the colloidal gold curve and, in 90 per cent of the cases, a positive Wassermann reaction are observed. In some instances there may be a considerable rise in temperature. The history, the associated neurologic findings, especially the pupillary changes in cases in which the involvement is latent, and the results of examination of the spinal fluid and of the blood establish the diagnosis.

6. Protective meningitis.

7. Otitic hydrocephalus.

The last two conditions are discussed under separate captions.

Prognosis: The outlook in bacterial meningitis of otorhinogenic origin was almost uniformly hopeless until the advent of sulfanilamide and related products. Even at this early date it has been established that the sulfanilamide compounds are instrumental in yielding recovery in a considerable number of cases.[19]

Treatment: At the time of this presentation there are but two direct effective methods of attack: chemotherapy and eradication of foci of infection.[20]

ABSCESS OF THE BRAIN

Intracerebral and intracerebellar abscesses are most commonly caused by infections of the temporal bone and paranasal sinuses. Table 4 indicates the relative frequency of the various causes of abscess of the brain as reported by other workers. Table 5 represents cases collected in my series.

It is obvious that disease of the temporal bone is the most frequent source of abscess of the brain. There were 23 abscesses in the temporal lobe, 4 in the cerebellum and 1 each in the frontal and the parietal lobe.

Sixteen abscesses of the brain were of rhinogenic origin. They were all in the frontal lobe. Fourteen of them were due to disease of the frontal sinus, alone or in combination with disease of other sinuses. One was ascribed to ethmoiditis and another to ethmosphenoid osteomyelitis. One abscess of the frontal lobe was due to disease of the temporal bone.

19. Neal, J. B., and Appelbaum, E.: Experience with Sulfanilamide in Meningitis, Am. J. M. Sc. **195**:175, 1938.

20. Schenck.[11] Neal and Appelbaum.[19]

Pathology and Pathogenesis.—The life history of an abscess of the brain has been well described by Courville [21] as follows:

Infection which has reached the pia mater from an adjacent focus usually gains entrance to the brain by way of (*a*) an entering artery, (*b*) an emergent vein, or

TABLE 4.—*Analysis of the Causes of Abscess of the Brain in 216 Cases*

	Evans, W.: Lancet **1**: 1231 and 1289, 1931	Parker, H. L.: Am. J. M. Sc. **180**: 699, 1930
I. Direct extension		
1. Otitis media and mastoiditis	109	
2. Suppuration in nose and accessory sinuses	12	
3. Trauma of skull	8	
4. Invasion of skull by malignant growth	2	
Total	131	
II. Hematogenous spread (metastatic)*		
1. Intrathoracic suppuration	22	14
2. Extrathoracic suppuration	26	6
Totals	48	20
III. Source of infection doubtful or unknown	17	

* Osteomyelitis and cystopyelitis were the most common initial lesions in this group. General sepsis occurred in the majority of cases.

TABLE 5.—*Abscess of the Brain Secondary to Infections of the Temporal Bone and Paranasal Sinuses*

A

	Number of Cases	Recoveries	Deaths	Mortality, Percentage
Abscess of frontal lobe	17	3	14	82
Abscess of temporal lobe	23	6	17	74
Abscess of parietal lobe	2	0	2	100
Cerebellar abscess	4	1	3	75
Total	46	10	36	
Average				83

B

	Abscess of Frontal Lobe	Abscess of Temporal Lobe	Abscess of Parietal Lobe	Cerebellar Abscess	Total
Disease of temporal bone					
Acute	..	8	..	1	9
Chronic	1	15	1	3	20
Disease of frontal sinus					
Acute	9	..	..	..	9
Chronic	5	..	1	..	6
Chronic ethmoiditis	1	..	..	..	1
Chronic ethmosphenoiditis	1	..	..	..	1
Total	17	23	2	4	46

(*c*) the perivascular spaces. Because of its more abundant blood supply, the cortex does not yield to infection which extends into the white matter. The first stage of the abscess may be an infarct in one case (arterial occlusion) or hemorrhagic softening in another (venous thrombosis). The type of this primary lesion probably predisposes to the type of abscess which is to develop. As infection

21. Courville, C. B.: Pathology of the Central Nervous System: A Study Based upon a Survey of Lesions Found in a Series of Fifteen Thousand Autopsies, Mountain View, Calif., Pacific Press Publishing Association, 1937.

spreads in the decadent tissues, the clinical symptoms are those of encephalitis. This process continues for three to five days, when an equilibrium is reached between the infection and the reaction of the enveloping brain. The regional blood vessels throw off a heavy exudation of leucocytes, which constitutes the first line of defense. Behind this wall the blood vessels consolidate the position by increasing in size and number. There is proliferation of the connective tissue cells of their adventitia. Thus a "capsule" is formed, which limits, at least temporarily, the size of the abscess.

From this time on, it is a struggle between the local reacting elements and the organisms within the abscess, between the capsule (field fortifications) and leucocytes (defending troops) on the one hand and invading bacteria (invaders) on the other. If the capsule yields at any point to the increasing intracapsular tension, the "break through" may result in an overwhelming extension into the surrounding white matter (spreading encephalitis) or may be again enveloped by a second capsule ("daughter" abscess). These eventualities are accompanied by a recurrence of signs of encephalitis.

Many practical problems concerning the histologic picture and especially the formation of the capsule in the abscess of the brain have received the attention of many investigators. Alpers,[22] after an exhaustive review of the literature and a histologic study of 26 cases, arrived at the following formulations: An abscess of the brain is composed of four layers from within outward: necrotic zone, reactive zone, fibrous zone (capsule) and encephalitic zone. He stressed the fact that an encephalitic zone often exists even in an abscess of long duration. The time required for the formation of the capsule depends on variable factors, of which the most important are as follows: 1. The nature of the invading organisms. It is generally agreed that the aerobic organisms, especially the cocci, favor formation of a capsule, whereas the anaerobes do not. The virulence of the invading organism may also be a determining factor in the formation of a capsule. 2. The resistance of the host. This is particularly important when one considers associated chronic infection, acute infections in the air cavities and osteomyelitis. 3. The time of onset of the abscess. This is often difficult to determine because the onset may be insidious or masked by the severe systemic reactions of the associated disease of the temporal bone or sinus. Generally speaking, the older the abscess the greater the likelihood of encapsulation. The capsule forms in a few days in some cases, but in others the capsule is not formed after as many as eleven months. Alpers concluded that the optimum time for formation of a capsule is three to four weeks.

Clinical Types.—The pathologic variability of abscess of the brain is reflected in the complicated clinical classification, into four types (Oppenheim [23]).

22. Alpers, B. J.: Abscess of the Brain: Relation of the Histologic to the Clinical Features, Arch. Otolaryng. **29**:199 (Feb.) 1939.

23. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, Berlin, S. Karger, 1923.

In the acute fulminating type the onset is sudden; rapidly spreading encephalitis results, and the patient succumbs quickly, long before encapsulation can occur.

In the subacute type, which is the one most frequently encountered, the abscess passes through three or four stages, each of which has its characteristic symptoms. The first stage is that of the onset of the cerebral infection, or the stage of acute encephalitis. The second is a latent stage. The third stage is that of cerebral suppuration, followed by the fourth stage, that of encapsulation. The time from the beginning of the infection to encapsulation is variously estimated to be four to six weeks, although a longer period may elapse.

With the latent type, the symptoms are vague or so obscure as to escape the notice of the attending otologist. Diagnosis is usually made at autopsy.

A fourth type of clinical evolution of abscess of the brain is like that of a tumor. The infection is of low virulence, and the abscess produces symptoms simulating those of a tumor, growing in the same region and at the same rate. Such an abscess is usually well encapsulated and is often enucleated en masse by the neurosurgeon.

The pathways of invasion are enumerated in a previous section, and their description is amplified in the discussion of the various groups.

Abscess of the Temporal Lobe.—The usual cause of abscess of the temporal temporosphenoid lobe is infection of the temporal bone. In rare instances the temporal lobe may be invaded from rhinogenic infection in one of three ways: (*a*) by lateral extension from the sphenoid sinus, (*b*) by osteomyelitis of the greater wing of the sphenoid from maxillary sinusitis and (*c*) from widespread pachymeningitis interna from frontal sinusitis.

The temporal lobe may be invaded by an infection of the temporal bone by extension (1) from the antrum through the tegmen tympani, (2) by way of the superior semicircular canal, (3) by way of the zygomatic cells, (4) by retrograde extension along venous channels and (5) by implantation of an exploring needle (Courville [21]).

Abscess of the temporal lobe may arise from acute or chronic infection, but it results most frequently from chronic infection with acute exacerbation.

Of the 23 abscesses of the temporal lobe in this series, 8 were due to acute disease of the temporal bone. Of the 8, 5 were on the left side and 3 on the right, and all but 2 were fatal. Fifteen were due to chronic disease of the temporal bone, with or without acute exacerbations. Of the 15, 9 were on the left side and 6 on the right, and all but 4 were fatal.

Of the 23 abscesses, 1 was associated with osteomyelitis of the zygoma; 3 with extradural abscess, 2 with subdural abscess and 3 with

thrombosis of the lateral sinus. Of the 17 fatal abscesses, the patient died of terminal meningitis with 11, of cerebral compression with 4 and of septicemia with 3.

Symptoms: The initial chill, usually described in all textbooks, is of little diagnostic value. In some cases it is possible to elicit a history of "grip" days or weeks prior to the onset of the more severe symptoms. Some of the "grippy" sensations may indicate the period of intracranial invasion. The analysis of this series did not yield a history of an "initial chill." I interviewed 15 otologists in Philadelphia, and not one of them could recall an "initial chill" with abscess of the temporal lobe.

TABLE 6.—*Analysis of Symptoms of Abscess of the Brain*

	Abscess of Temporal Lobe (23 Cases)	Abscess of Frontal Lobe (17 Cases)
Initial chill	None	17
Headache	23	17
Dizziness	8	6
Vomiting	18	7
Mental changes	20	12
Rise in temperature	Slight and in only a small number	Variable temperature in all cases
Slow pulse	19	5
Papilledema	In 8 of 18 patients examined	9
Hemianopic defects	In 11 of 17 patients examined	None
Oculomotor palsy	3	3
Palsy of abducens nerve	4	4
Trigeminal pain	2	No definite data
Palsy of central facial nerve	20	12
Aphasia	In 8 of the 14 lesions on the left side	1
Hemiparesis	6	7
Jacksonian convulsions	1	4
Generalized convulsions	2	4
Increased spinal fluid pressure	In 7 of 17 cases in which data were recorded	In 7 of 9 cases in which data were recorded
Pleocytosis	6 (15 to 100 cells)	9 (15 to 250 cells)
Roentgen abnormalities	None	Shift of pineal body in 1 case
Edema of orbital region	..	5
Exophthalmos	..	6
Meningeal irritation	..	4

Headache was present in all the 23 cases. The degree varied, but in most cases the headache was severe. It was generalized in most instances. In a few it was lateralized, and in 3 there was tenderness to percussion over the temporoparietal region.

Dizziness occurred in 8 cases. It was not vertiginous and was influenced by changes in posture of the head or sudden effort.

There was a history of vomiting in the early stages of the disease in 14 cases. It occurred in the early or the later stages in 18 cases. It may be the earliest manifestation and may be so conspicuous as to obscure the whole clinical picture, as in case 2.

The mental dulness associated with abscess of the brain is well known. The associated somnolence and restlessness should not be con-

fused with the aphasia so common with abscesses of the temporal lobe. Mental changes were observed in 20 cases. In the other 3 the mental state is not clearly recorded.

The temperature in the majority of cases was normal or only slightly elevated unless the abscess was associated with other processes (acute otitis, mastoiditis, sinus thrombosis).

The pulse was slow in 19 of the 23 cases. The rate varied between 46 and 84.

No significant conclusions could be derived from the blood pressure. In most cases frequent blood pressure readings had not been recorded. In some cases a variation of pulse pressure between 20 and 40 mm. of mercury appeared.

Examination of the fundi was recorded in 18 cases. In 8 of these papilledema was noted, varying from 1 to 4 diopters.

Fields of vision were recorded in 17 cases. In 11 of these some hemianopic defects were seen contralateral to the side of the lesion.

Homolateral oculomotor palsy was observed in 3 cases. Palsy of the abducens nerve was recorded in 4 cases. Pain referable to the trigeminal nerve on the side of the lesion was recorded in 2 cases. Central palsy of the contralateral facial nerve occurred in 20 of the 23 cases. Hemiparesis of a variable degree was observed in 6 of the 23 cases.

Sensory and nominal aphasia were recorded in 8 of the 14 cases of a lesion on the left side.

Jacksonian convulsions occurred once in the 23 cases. Generalized convulsions occurred in 2 cases.

Spinal fluid pressure was recorded in 17 of the 23 cases. In 7 cases it was definitely elevated.

The cell content of the spinal fluid was recorded in only 15 cases. In 6 of these slight pleocytosis appeared, the cell count varying from 15 to 100.

Roentgen studies in this series did not give evidence of increased intracranial pressure, calcification or pineal shift.

Vestibular studies were performed and aided in excluding the posterior fossa as the site of the lesion and occasionally in lateralizing it.

An analysis of the symptoms of the abscesses of the temporal lobe in this series reveals the findings in the order of frequency to be as follows: Headache, mental dulness, palsy of the contralateral facial nerve, bradycardia, vomiting, hemianopic defects of the fields of vision, aphasia, papilledema, dizziness, increase in spinal fluid pressure, increase of cells in the spinal fluid and hemiparesis. Other findings are so infrequent as to be of little diagnostic value. Most of the symptoms and signs recorded are merely indications of increased intracranial pressure and of meningeal irritation. The findings of real localizing value are

central contralateral palsy of the facial nerve, hemianopic defects of the fields of vision and aphasia.

Diagnosis and Treatment: The diagnosis of abscess of the temporal lobe is usually difficult. In the early and the acute stage it is confused most frequently with (*a*) extradural and subdural abscesses, which can be diagnosed only by exploration; (*b*) diffuse bacterial leptomeningitis and protective meningitis, and (*c*) thrombosis of the lateral sinus. In the subacute and the chronic stage or in any stage, it may be confused with (*a*) cerebellar abscess, (*b*) tumor of the brain, (*c*) localized encephalitis or (*d*) arachnoiditis and otitic hydrocephalus.

The treatment of abscess of the temporal lobe will be discussed at the end of the section on abscesses of the brain.

Abscess of the Frontal Lobe.—Abscesses of the frontal lobe are usually due to sinusitis. Yet Courville and Rosenvold,[5] who made an exhaustive review of the literature, concluded:

> Abscess of the brain is a relatively rare complication of sinusitis, as compared with other rhinogenic complications and with abscess from other sources. . . . In our series of 15,000 autopsies are recorded 7 cases of rhinogenic abscess of the brain, as compared with 47 in which such an abscess was of otitic origin. . . . In a series of 363 cases of fatal disease observed at the Manhattan Eye, Ear, Nose and Throat Hospital, Eggston[24] found reports of 8 cases in which abscess of the brain was secondary to sinusitis.

The most frequent cause of rhinogenic abscess of the brain is suppurative frontal sinusitis, and the most common location for such an abscess is in the frontal lobe. However, frontal sinusitis may produce abscesses in locations other than the frontal lobe, and abscess of the frontal lobe may be due to conditions other than frontal sinusitis.

Frontal abscess secondary to frontal sinusitis (*a*) may be due to venous thrombophlebitis, which is probably the most common mode, (*b*) may develop by contiguity, with local necrosis of bone (osteomyelitis) and of the dura and local adhesions to the brain and intracerebral involvement, and (*c*) may result from subdural infection, in the form of either diffuse pachymeningitis interna or frank subdural abscess.

It is important to remember that occasionally an abscess of the frontal lobe may make its clinical appearance when the original sinal infection has completely disappeared, as in case 1 in this series. It is not at all impossible that healed sinal infection plays an etiologic role in abscesses of the brain of "undetermined" cause (Yaskin, Grant and Groff[25]), i. e., undetermined at the time the patient presents himself with an abscess of the brain, long after the original infection has cleared up.

---

24. Eggston, A. A.: Pathways of Infection in Suppurative Meningitis, Ann. Otol., Rhin. & Laryng. **43**:672, 1934.

25. Yaskin, J. C.; Grant, F. C., and Groff, R. A.: Brain Abscess of Undetermined Etiology, Ann. Surg. **107**:492, 1938.

Frontal abscesses may be solitary or multiple, and they are frequently multilocular.

Less commonly abscesses of the frontal lobe are due to ethmoiditis (Dreyfuss,[26] Toti,[27] Hagerup,[28] Maxwell[29]), sphenoiditis (Kramer,[30] Lischkoff[31]) or inflammation of the maxillary sinus (Dreyfuss[26]). The degree of encapsulation and associated encephalitis follows the same general rules as in abscesses in other parts of the brain.

While most abscesses of the frontal lobe secondary to frontal sinusitis occur on the same side as the predominantly involved sinus, in some cases the contralateral lobe may be the seat of the abscess, as in case 10 of this series. Contralateral involvement is explained by (*a*) deviation of the internal septum, which permits the spread of the infection through the posterior wall of one sinus into the frontal lobe of the opposite side and (*b*) venous thrombophlebitis, the infection traveling by way of the superior longitudinal sinus and its afferent veins.

In this series there were 17 patients with abscess of the frontal lobe, of whom only 3 survived.

In 14 of the 17 cases the abscess originated in disease of the frontal sinuses. In 9 of the 14 cases it was classified as due to *acute* or *subacute frontal sinusitis.* In at least 4 there was a history of diving and swimming prior to the onset of the acute symptoms. In 3 of the 9 cases clinical and roentgen evidences of osteomyelitis of the frontal and adjacent bones appeared. In 2 of the cases there was a history of definite cranial trauma. In 5 of the 14 cases the abscess was classified as due to chronic frontal sinusitis. In 3 of these clinical and roentgen evidences of osteomyelitis of the frontal bone appeared. In at least 5 of the 14 cases there was evidence of considerable involvement of the other paranasal sinuses.

In 1 of the 17 the abscess was ascribed to ethmoiditis, in 1 to sphenoethmoid osteomyelitis and in 1 to chronic disease of the temporal bone.

---

26. Dreyfuss, R.: Rhinogene Gehirnaffektionen, Internat. Zentralbl. f. Ohrenh. **6**:103, 1907.

27. Toti, E.: Kasuistik der intrakraniellen Komplikationen akuter und chronischer Nebenhöhlenerkrankungen seit den Arbeiten von Gerber und Dreyfuss bis zum Jahre 1923, Ztschr. f. Hals-, Nasen- u. Ohrenh. **7**:41, 1923.

28. Hagerup, G.: Rhinogenous Intracranial Complications: On the Basis of Material of the Department for Diseases of the Ear of Municipal Hospital, Copenhagen, Acta oto-laryng. **24**:321, 1936.

29. Maxwell, D.: Analysis of the Statistical Tables for the Years 1907 to 1926 Inclusive, Covering a Period of Twenty Years, J. Laryng. & Otol. **44**:105, 1929.

30. Kramer, R.: Intracranial Complications Following Sphenoid Infections, Laryngoscope **39**:573, 1929.

31. Lischkoff, M. A.: Latent Brain Abscess Secondary to Sphenoid Sinusitis with Case Report, South. M. J. **28**:700, 1935.

Autopsy was performed in 7 of the 17 cases, and the following conditions noted: septic meningitis in all 7 cases, osteomyelitis of the frontal bone in 2 cases, multiple abscesses in 4 cases, subtentorial abscess in 1 case and cerebral hemorrhage in 1 case.

In 1 case the abscess of the frontal lobe developed contralateral to the sinal infection (case 10).

Symptoms: The mode of onset was as follows: In all cases there was a history of acute or chronic sinal infection. In 4 cases there was a history of diving and swimming prior to the onset of acute symptoms, and in 2 there was a history of cranial trauma several months preceding the development of cerebral manifestations. In all but 1 of the cases evidences of active sinusitis were seen at the time of the development of signs of involvement of the brain. The 1 case is of considerable interest and is reported (case 1).

In all cases there was a history of chill, grip or evidence of a septic process. This contrasts with the clinical course of otitic abscess.

Headache was a prominent symptom in all the 17 cases. It was frequently lateralized, deep and accompanied by tenderness to percussion.

Dizziness was recorded in 6 of the cases. It was of the postural and rather indefinite type (sepsis, asthenia).

Vomiting occurred at one time or another in 7 cases. It was prominent and associated with severe headache in younger subjects, especially in case 1.

The mental state varied considerably, especially depending on the severity of the associated osteomyelitis and other sepsis. Delirium was present in 9 cases and elation in 3 cases; mental lucidity was a conspicuous finding in 2 cases.

A slight rise in temperature (to 99 or 100 F.) was present in all cases. Considerable fever (temperatures of 102 to 105 F.) was present in cases of abscess associated with osteomyelitis or other evidences of sepsis.

Bradycardia (pulse rates of 44 to 68) was recorded in 5 cases. In the majority of cases the pulse was slow in relation to the temperature.

No significant deductions could be derived from the meager blood pressure records.

Edema of the orbital region on the side of abscess was found in 5 of the 17 cases.

Exophthalmos on the side of the abscess was recorded in 6 cases.

Papilledema of varying degrees (1 to 6 diopters) was found in 9 of the 17 cases.

Defects in the fields of vision of definite value in localizing the lesion were not found.

Oculomotor palsy was recorded in 3 cases. Palsy of the abducens nerve was recorded in 4 cases. Central palsy of the facial nerve contralateral to the lesion was recorded in 12 of the 17 cases. Hemiparesis of a variable degree was observed in 7 of the 17 cases.

Mixed aphasia was recorded in only 1 case.

Generalized or jacksonian convulsions occurred in 4 of the 17 cases.

Meningeal irritation prior to onset of septic meningitis was recorded in 4 of the 17 cases.

Spinal Fluid pressure was recorded in 9 of the 17 cases, in 7 of which it was definitely elevated.

The cell count of the spinal fluid was recorded in 9 cases in all of which some pleocytosis (15 to 250 cells) appeared.

Roentgen studies showed no evidence of an increase in intracranial pressure or direct evidence of localizing value except in 1 case (case 10), in which a pineal shift localized the lesion contralateral to the sinal infection.

Vestibular studies proved of no definite value in the few cases in which the tests were performed.

Diagnosis and Treatment: On the basis of this study the diagnosis of abscess of the frontal lobe depends on the following common factors: (*a*) a history of or the existence of sinusitis, especially frontal sinusitis; (*b*) evidences of increased intracranial pressure, of which the most frequent are headache, vomiting, increased intraspinal pressure, papilledema and bradycardia; (*c*) evidences of focal disease of the brain, the most frequent being mental changes, central palsy of the contralateral facial nerve, hemiparesis, convulsions and, in 1 case each, pineal shift and aphasia, and (*d*) local symptoms, including exophthalmos, orbital edema, oculomotor palsy and possibly palsy of the abducens nerve.

Abscess of the frontal lobe may be confused with (*a*) osteomyelitis of the frontal or adjacent bones, (*b*) subdural or extradural abscess, (*c*) protective or bacterial meningitis, (*d*) localized nonsuppurative encephalitis or meningoencephalitis, (*e*) arachnoiditis and (*f*) tumor of the brain. Differentiation is based on careful (and repeated) clinical study, roentgenograms of the skull and sinuses, studies of the spinal fluid and, most frequently, exploration.

The treatment is discussed at the end of the section on abscess of the brain.

Other Cerebral Abscesses.—Rarely, abscess of the parietal or the occipital lobe, single or multiple, follows infection of a sinus or temporal bone by continuity, but more commonly it develops through widespread thrombophlebitis or pachymeningitis interna. In this series there were 2 abscesses of the parietal lobe. One was due to frontal sinusitis and

was probably a "daughter" abscess of a frontal abscess. The other was due to subacute disease of the temporal bone. It was accurately localized by the presence of astereognosis, but the patient succumbed to meningitis.

Cerebellar Abscess.—Cerebellar abscess is much less common than cerebral. In the great majority of cases it results from chronic disease of the temporal bone, and in only 15 per cent of cases does it follow acute processes. It is usually preceded by labyrinthitis, thrombosis of the lateral sinus or epidural abscess, the antecedent lesions indicating the pathways of invasion.

The diagnosis of a cerebellar abscess is usually difficult. Of utmost importance is a history of labyrinthitis or the finding at a previous operation of thrombosis of the lateral sinus, of an epidural abscess or of labyrinthitis or a labyrinthine fistula. The clinical picture is frequently masked by the presence of complicating conditions, such as thrombosis of the lateral sinus and labyrinthitis. The systemic reactions are not characteristic. They are those of the associated sepsis and vary considerably. The changes in the spinal fluid are the same as in other abscesses of the brain. Because the abscess is in the infratentorial region, evidences of increased intracranial pressure may be marked: headache, vomiting, bradycardia, respiratory irregularity, early and often marked papilledema without hemianopic defects and increased intraspinal pressure. The focal cerebellar signs are often difficult to elicit because of inability on the part of the patient to cooperate and because of the actual difficulty in evaluating the symptoms. These points are emphasized by an exhaustive investigation by Ramadier and his co-workers.[2] Hypermetria, asynergia, adiadokokinesis, past pointing, vertigo, nystagmus and spontaneous falling, when present, are of much value. The vestibular findings are at times important but must be critically evaluated. A much neglected sign is *hypotonia,* which may be elicited even in a stuporous patient but is often mistaken for hemiparesis. Hemiparesis is contralateral to the ear involved, while hypotonia and the other focal cerebellar signs are ipsilateral with the cerebellar lesion. The diagnosis is aided by the absence of focal cerebral signs, such as hemianopic defects and aphasia, although dysarthria of cerebellar origin is occasionally observed. The diagnosis is rendered more difficult by variability of symptoms from day to day, requiring repeated careful examinations and comparisons.

The following conditions are to be differentiated from cerebellar abscess:

1. Abscess of the temporal lobe, by previous operative findings, by the presence of hemianopic defects, aphasia, central palsy of the facial nerve and other evidences of cerebral involvement and by the absence of focal cerebellar signs and of early or marked papilledema.

2. Labyrinthitis, by the presence of severe vertigo, vestibular nystagmus (Sommer and Yaskin [32]) and positive reactions to Bárány tests and by the absence of evidence of increased intracranial pressure and focal cerebellar signs.

3. Cerebellar tumor, by the absence of aural suppuration and its complications, by the slower development of the evidences of increased intracranial pressure and of the focal cerebellar signs and by consistently negative findings in the cytologic study of the spinal fluid.

4. Pseudotumor of the posterior fossa, by results of examination of the spinal fluid, by the relief of symptoms by lumbar or ventricular puncture or by operative findings.

5. Meningitis, by examination of the spinal fluid and by the absence of focal cerebellar signs.

In this series there were, in all, 4 cerebellar abscesses, of which 3 (2 on the left and 1 on the right) were associated with chronic disease of the temporal bone and 1 (on the left) with acute mastoiditis. Three patients died; 1 recovered after operation. Two abscesses were associated with extradural as well as with intracerebellar collections of pus, and 1 patient had thrombosis of the lateral sinus.

In 3 of the cases clinical evidences of meningeal irritation appeared. In all cases increased intracranial pressure was manifested, by headache, vomiting, slowness of the pulse, increased intraspinal pressure and papilledema. In 1 case respiratory irregularity appeared. The mental state was dull, but not as pronouncedly as with abscesses of the temporal lobe. Nystagmus was present in 3 of the 4 cases. Hypotonia, ipsilateral with the abscess, was found in 2 cases. Hypermetria, dyssynergia and adiadokokinesis were observed in 3 of the 4 cases. Hemiparesis on the side contralateral to the lesion was found in 1 case, and in another instance there was associated peripheral palsy of the facial nerve.

Treatment of Abscess of the Brain.—The treatment of abscess of the brain remains unsatisfactory, as is best proved by the high mortality (Grant [33]). The difficulties of the treatment are traceable to inherent pathologic factors, to diagnostic difficulties and to difficulties of surgical approach.

There is general agreement on at least three factors, namely, eradication of foci in the temporal bone and paranasal sinuses, free use of sulfanilamide and related chemotherapeutic agents and waiting until the abscess of the brain is encapsulated. The first two factors offer no serious obstacles, while the last is beset with numerous difficulties.

32. Sommer, I., and Yaskin, J. C.: Spontaneous Nystagmus: Some Practical Clinical Features, Especially Its Occurrence with Diplopia, Arch. Ophth. **2**:57 (July) 1929.

33. Grant, F. C.: Prognosis for Abscess of the Brain, Arch. Neurol. & Psychiat. **40**:603 (Sept.) 1938.

The ideal plan is to wait until the abscess is completely encapsulated, so that a minimum of encephalitis surrounds it, and then to operate by one of the four recognized methods: the closed method, the open method, extirpation in toto and tapping (King [34]). The most satisfactory results are obtained when the well encapsulated abscess is removed en bloc (Vincent and David [35]). Two cases in which this was done came under my observation and in both cases the preoperative diagnosis was tumor of the brain (Yaskin, Grant and Groff [25]). Practically, however, there are many obstacles to this ideal plan. In the first place, there is frequently serious doubt as to the existence of an abscess, and yet, by reason of a marked increase in intracranial pressure or serious focal disease of the brain, surgical intervention becomes necessary. In the second place, it is frequently impossible to determine when encapsulation takes place, and, furthermore, some abscesses never encapsulate ("Pathology and Pathogenesis"). It is generally agreed that encapsulation takes place in four to six weeks from the time of the beginning of the suppurative encephalitis. It is often impossible to state with certainty when the abscess began to form, and the calculations are therefore inaccurate. It is claimed that when the spinal fluid pressure and the papilledema become stationary (Lillie [36]) and the spinal fluid cell count shows a reduction of neutrophils and only some lymphocytes (Woltman [37]) encapsulation has taken place and the time is ideal for operative intervention.

Neurosurgeons and some otorhinologists are of the opinion that an abscess of the brain should be operated on through a clean field and not through the infected channels in the temporal bone or paranasal sinuses (Adson and Craig [38]). Some otorhinologists, however, believe in attempting to trace the abscess through the pathways of infection responsible for it. This is probably unavoidable in cases of abscess of the frontal lobe associated with osteomyelitis of the bones of the skull. Many otorhinologists avoid opening the dura when they believe that the abscess of the brain is deeply situated or well encapsulated and prefer to turn the patient over to the neurosurgeon for exploration through a clean field.

(To Be Concluded)

34. King, J. E. J.: Brain Abscess, Ann. Surg. **103**:647, 1936.

35. Vincent, C., and David, M.: Sur l'ablation en masse sans drainage des abcès subaigus des hémisphères cérébraux, Rev. d'oto-neuro-opht. **14**:1, 1936.

36. Lillie, W. L.: The Clinical Significance of Choked Disk Produced by Brain Abscess, Surg., Gynec. & Obst. **47**:405, 1928.

37. Woltman, H. W.: Spinal Fluid Cell Count and Encapsulation of Brain Abscess: Attempt to Correlate These Factors, and to Determine Optimal Time for Drainage, J. A. M. A. **100**:720 (March 11) 1933.

38. Adson, A. W., and Craig, W. McK.: The Mechanical Management of Brain Abscess, Ann. Surg. **101**:7, 1935.

ORIGIN AND DISTRIBUTION OF AIR CELLS IN THE TEMPORAL BONE

OBSERVATIONS ON SPECIMENS FROM TWENTY-SEVEN INFANTS AND SIXTY-NINE HUMAN FETUSES

T. H. BAST, PH.D.
AND
H. B. FORESTER, PH.D.
MADISON, WIS.

The recent interest in pneumatization and suppuration of the petrous portion of the temporal bone is shown by the number of articles on the subject which have appeared since 1930. Most of these articles are clinical and only a few anatomic in nature. Gross, roentgen and corrosion studies represent the anatomic methods employed in the majority of cases. These methods, as pointed out by Guild,[1] Jones [2] and Anson, Wilson and Gaardsmoe,[3] are not always accurate and should be supplemented with a study of serial histologic sections. The demand for observations on pneumatization of the petrous bone based on serial histologic sections warrants this report.

REVIEW OF THE LITERATURE

In the study of air cells various methods have been employed. Bezold [4] and Siebenmann [5] employed the corrosion method.

From the Department of Anatomy, University of Wisconsin.

The collection and preparation of the material used in this study were supported by a grant from the Research Council of the American Otological Society. The actual study and analysis of this material were made possible by a grant from the Wisconsin Alumni Research Foundation.

This is a preliminary report. Further studies on air cells by other methods and on older bones are being made by one of us at present.

1. Guild, S. R.: Normal and Pathological Anatomy of the Petrous Pyramid, Tr. Am. Otol. Soc. **25**:165-192, 1935.

2. Jones, M. F.: Normal and Pathological Anatomy of the Petrous Pyramid, Tr. Am. Otol. Soc. **25**:192-205, 1935.

3. Anson, B. J.; Wilson, J. G., and Gaardsmoe, J. P.: Air Cells of Petrous Portion of Temporal Bone in a Child Four and One-Half Years Old, Arch. Otolaryng. **27**:588-605 (May) 1938.

4. Bezold, F.: Die Corrosions-Anatomie des Ohres, Munich, Literar.-Artist. Anstalt., 1882.

5. Siebenmann, F.: Mittelohr und Labyrinth, in von Bardeleben, K.: Handbuch der Anatomie des Menschen, Jena, Gustav Fisher, 1887, vol. 5, pt. 2.

Another method was to make dissections of saw cut sections of petrous bones. Using this gross method, Belinoff and Balan [6] studied petrous bones and reported pneumatization of the petrous pyramid in 78 per cent of cases. Using the roentgen and the gross method, Myerson, Rubin and Gilbert [7] studied 200 temporal bones obtained from 100 skulls ranging in age from 7 fetal months to 87 years. They found 11 per cent of the pyramids and 38 per cent of the mastoids to be pneumatic, while the rest were nonpneumatic. Kraus [8] on the basis of roentgen studies of 604 persons reported about 24 per cent with the petrous apex pneumatized. Fowler Jr.[9] made roentgenograms of petrous bones and then made serial histologic sections to check with his roentgen findings. His results indicate that the roentgen method is not dependable for the determination of pneumatization. Glick,[10] who studied serial sections of 42 petrous bones from 27 persons, found 12 pneumatic pyramids. No pneumatization was found in 8 infants ranging in age from 2 months to 1 year. Friesner, Druss, Rosenwasser and Rosen [11] studied 24 petrous bones, making sections up to the tip of the apex of 20, and found the apex pneumatic in 5 per cent of 20 bones, mixed in 30 per cent and diploic in 65 per cent, the mastoid pneumatic in 50 per cent of 24 bones, mixed in 37.5 per cent and diploic in 12.5 per cent and the pyramid pneumatic in 16.67 per cent of 24 bones, mixed in 75 per cent and diploic in 8.33 per cent. Jones [2] and Guild,[1] who also studied histologic sections, pointed out the importance of the study of large numbers of serial sections in order to arrive at accurate percentages. Guild [1] laid emphasis on the outlet of the petrous air cells and on the specific location of specific groups of these cells. Wilson,[12] in his study of histologic sections of petrous bones, concluded that apical pneumatization is less frequent than is commonly believed and suggested that the study of diseased bones may lead to erroneous conclusions.

6. Belinoff, F., and Balan, M.: Ueber den Bau der Pyramidenspitze, Monatschr. f. Ohrenh. **64**:1185-1188 (Oct.) 1930.

7. Myerson, M. C.; Rubin, H., and Gilbert, J. G.: Anatomic Studies of the Petrous Portion of the Temporal Bone, Arch. Otolaryng. **20**:193-210 (Aug.) 1934.

8. Kraus, L.: Die Pyramidenspitzenpneumatisation im Röntgenbild, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **128**:307-338 (May 11) 1931.

9. Fowler, E. P., Jr.: The Correlation of X-Ray and Pathologic Findings in Suppurations of the Petrous Pyramid, Tr. Am. Otol. Soc. **25**:215-228, 1935.

10. Glick, H. N.: Microscopic Observations of the Petrous Apex, Ann. Otol., Rhin. & Laryng. **42**:175-191 (March) 1933.

11. Friesner, I.; Druss, J. G.; Rosenwasser, H., and Rosen, S.: Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:659-688 (Dec.) 1935.

12. Wilson, J. G.: Anatomy and Pathology of Petrous Tip, Tr. Am. Otol. Soc. **25**:205-215, 1935.

In this connection it is of interest to note that many if not most of the studies just reported were based on suppurating or otherwise diseased petrous bones.

Anson, Wilson and Gaardsmoe [3] reported on a reconstruction of serial sections showing extensive pneumatization of the petrous bone of a 4½ year old child.

PURPOSE

A review of the literature shows that comprehensive accounts of pneumatization based on large numbers of petrous bones describe only gross, roentgen and corrosion studies. The accounts derived from histologic sections (a method which is conceded to be more accurate) are less complete and are based on small series or isolated cases, which give some information regarding frequency, degree and variation but little regarding the origin, development and grouping of air cells in a representative series of petrous pyramids. The purpose of this paper, therefore, is to present observations on origin, development, distribution, frequency and grouping of air cells as seen in serial histologic sections of a large number of human fetal and infant petrous bones. The relation of developing air cells to marrow spaces and the relation of apical and mastoid cells will be discussed.

MATERIAL AND METHODS

This report on air cells is based on the study of serial histologic sections of 39 temporal bones of 27 infants, ranging in age from full term to 6 years, and 97 temporal bones of 69 fetuses, ranging in age from 8 weeks to term. The bones were sectioned horizontally in their entirety with the exception of some of the older ones, from which part of either the mastoid or the apical region had been cut away. Each section for each series was projected for study at a magnification of 20 diameters or more. Detailed study of specific areas was made under a microscope. Drawings and photomicrographs were made of representative regions.

Anson, Wilson and Gaardsmoe [3] in a recent paper stated that the only reliable method of study is by reconstructions from serial sections. We are in agreement with them that reconstructions bring out relations which are hard to visualize from direct histologic observation of serial sections. In the face of this confession it may seem strange that we should give a report not entirely based on reconstructions. We have modeled some of the air cells, and photographs of one of the models are shown (figs. 25 and 26), but it is obvious that it would be an endless task to make reconstructions of serial sections of 130 temporal bones. In justification of this account with but limited reconstructions may we state that we have made numerous reconstructions of temporal bones at the laboratory of the department of anatomy of the University of Wisconsin during the past thirteen years? Some of the reconstructions of adult temporal bones, not included in this report, dealt specifically with pneumatization. This experience gives us a certain confidence in our ability to make a reasonably correct interpretation of serial sections of temporal bones and warrants our present report.

The results of our study are here discussed under the following headings:

Problem of Pneumatization
Origin and Development of the Middle Ear and Antrum
  Mastoid and Antral Air Cells
Origin and Distribution of Petrous Air Cells
  Antral Air Cells
  Carotid Air Cells
    Subtubal Group
    Postcarotid Group
    Precarotid Group
    Precochlear Group
  Supracochlear Air Cells
  Apical Air Cells
Outlet of Air Cells
Interrelation of Groups of Air Cells
Relation of Air Cells to Bone Marrow
Percentage of Pneumatization of Petrous Bone

PROBLEM OF PNEUMATIZATION

A review of the literature shows general agreement that the mastoid air cells, which are the most constant, are an extension of the tympanic cavity and its lining epithelium through the antrum into the mastoid bone. Regarding the time and method of pneumatization of the middle ear and antrum, little information is available, and regarding the time of formation of mastoid cells, there is no definite agreement. McMurrich [13] and Gray [14] stated that air cells in the mastoid region appear at or slightly before birth. Most other textbooks have stated that they are not present at birth but gradually begin to appear during the first two years of infancy. Still others have stated that they do not develop until the age of 2 years. Quain [15] said, in speaking of the mastoid process: "The process begins to appear in the second year, but the air-cells in it are not formed till near puberty."

Regarding the time of the appearance of apical air cells, no information is given in the literature. The method of formation of air cells is generally described as a resorption of the diploic bone and an invasion of epithelium.

This difference of opinion regarding the time of the appearance of the pneumatic spaces indicates that individual interpretation must play a considerable part in the conclusions reached. It must be remembered

13. McMurrich, J. P.: The Development of the Human Body, Philadelphia, P. Blakiston's Son & Co., 1923, p. 453.

14. Gray, H.: Anatomy of the Human Body, ed. 22, edited by W. H. Lewis, Philadelphia, Lea & Febiger, 1930, p. 138.

15. Quain, J.: Elements of Anatomy, edited by E. Schafer, J. Symington and T. H. Bryce, London, Longmans, Green & Co., 1915, vol. 4, pt. 1, p. 68.

that the temporal bone is growing and changing in structure to the time of puberty. The middle ear, of which the antrum and air cells are outgrowths, is likewise changing during this period.

For example, a certain air cell in an infant may become part of the middle ear or of the antrum in the adult. In other words, the middle ear and the antrum change during childhood and may incorporate some of the early formed air cells. This may result in a difference of interpretation. Then, too, there are marked differences in the structures of the middle ear and the antrum in different persons. Some middle ears reach their definitive shape and size early and have a fairly smooth contour. Others may have an irregular contour with outpocketings, which may continue to change for some time. In such instances it is often impossible to determine (at least in the earlier stages) whether the outpocketings represent permanent air sacs or irregularities of the middle ear. However, the method of formation is the same in either case.

ORIGIN AND DEVELOPMENT OF THE MIDDLE EAR AND ANTRUM

Pneumatization of the middle ear is produced by the invasion of the nasopharyngeal epithelium through the auditory tube (eustachian tube) into the connective tissue of the embryonic middle ear. This invasion of the auditory tube occurs early in embryonic life, as is shown in figure 1, which is a photomicrograph of a section through the ear of a 25 mm., or 8 week old, human fetus. At this stage the future bones of the middle ear are masses of precartilage. The middle ear is not yet outlined, but the connective tissue in this region is largely of the embryonic cellular type except for a small area between the stapes and the end of the auditory tube, where a dedifferentiation into loose mesenchymal tissue is taking place. The connective tissue is loosened throughout the region of the middle ear before the epithelium from the auditory tube invades it. In an embryo of 50 mm., or at the age of 11+ weeks, the future otic bones are masses of cartilage. The middle ear is well delimited and is filled with loose vascular connective tissue. The auditory tube has not penetrated much farther than in the 8 week stage. Pneumatization of the middle ear is slow and is not complete until late in fetal life. In a fetus of 24 weeks it involves only a small part of the middle ear and is still incomplete as late as the thirty-fourth week of intrauterine life, as may be seen from figure 8.

Before pneumatization of the middle ear has progressed far, the loose mesenchymal connective tissue of the middle ear tunnels toward the area which later will be the antrum. This process (arrow, fig. 4) occurs between the tympanic plate (*T. P.*) and the ossifying otic capsule. As they ossify laterally it expands between them into the area of the future antrum. In a 22 week fetus (fig. 5) it has reached the region

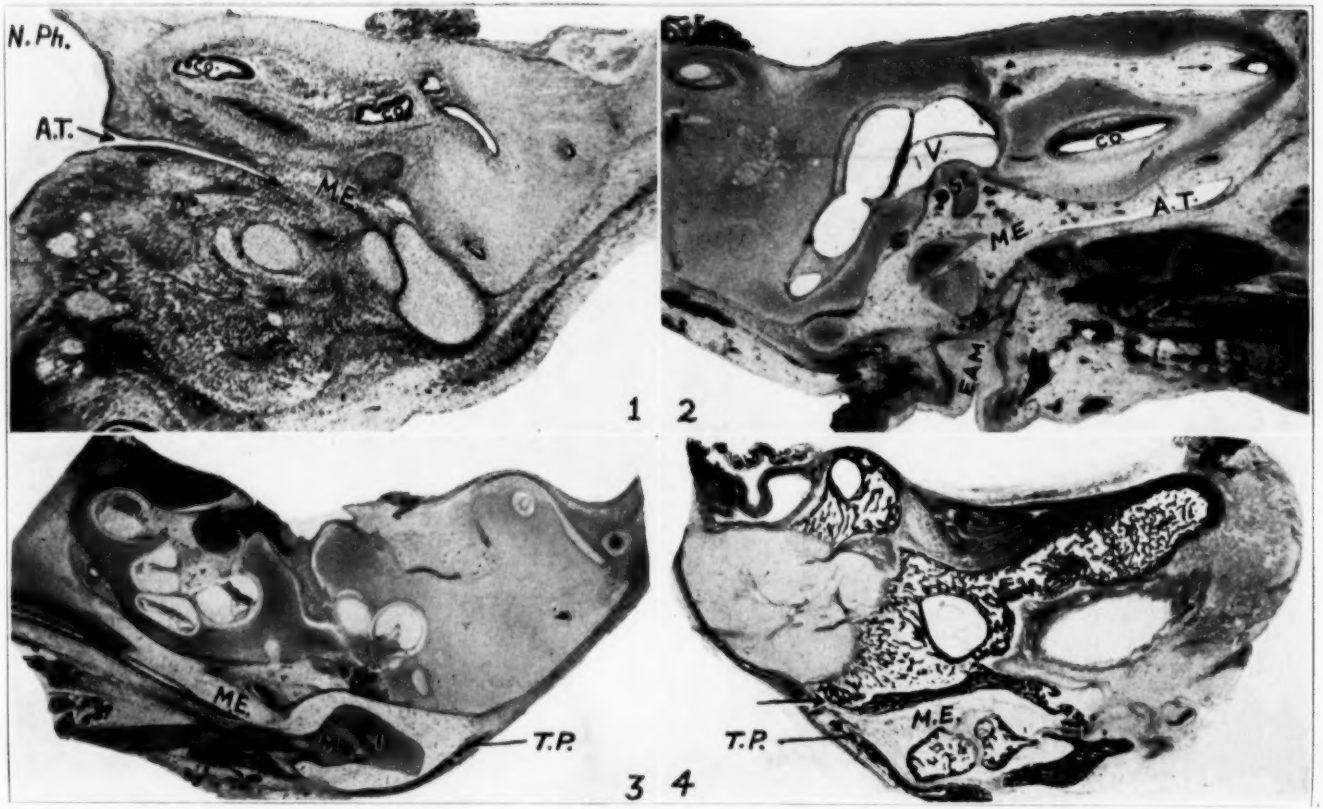

N. Ph.
A.T.
M.E.
1
IV.
CO.
ME
A.T.
EAM
2
M.E.
T.P.
3
T.P.
M.E.
4

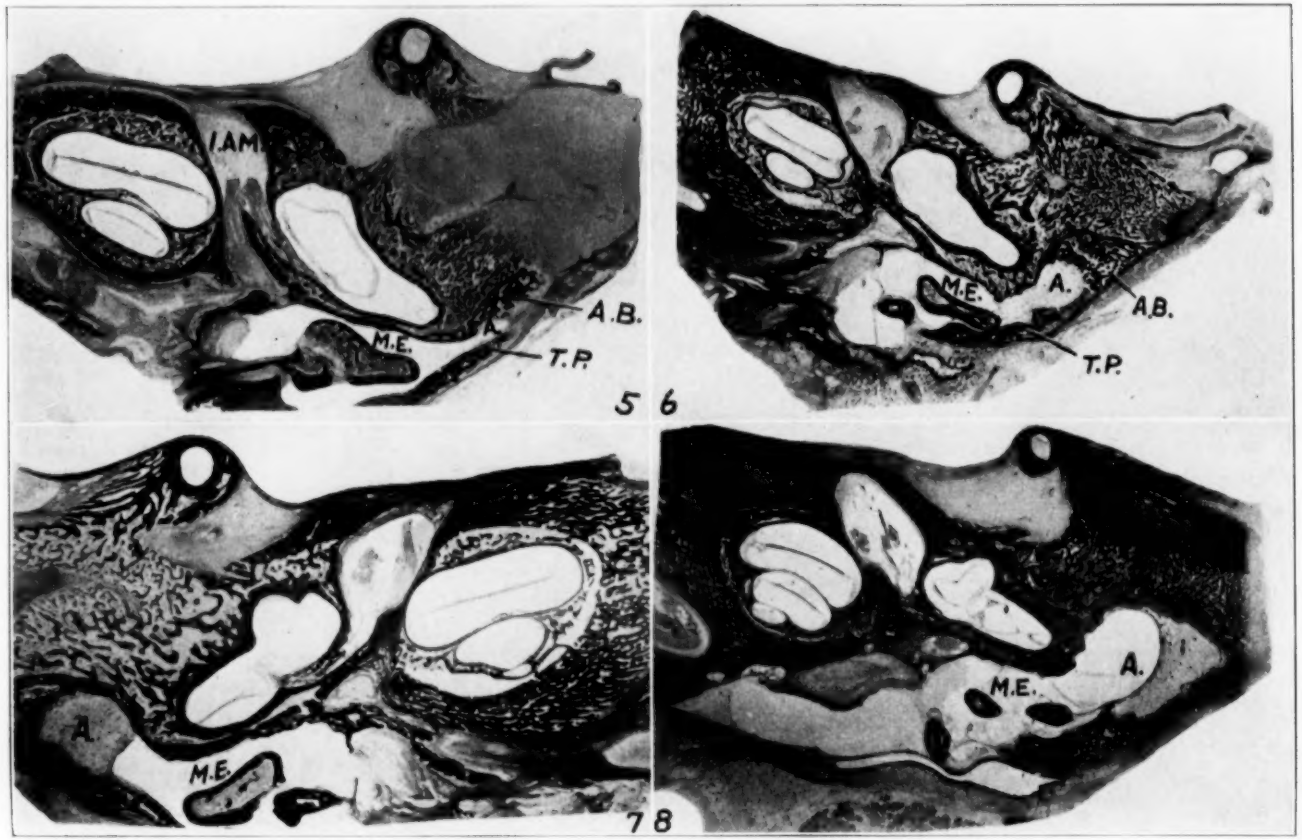
Fig. 1 (embryo 87, 25 mm. crown-rump length, age about 8 weeks).—Section through the left side of the head. The auditory (eustachian) tube extends from the nasopharynx to the region of the middle ear. The connective tissue of the middle ear is beginning to loosen. The bones of the middle ear exist as masses of early cartilage. The otic capsule is still in a precartilaginous stage.

Fig. 2 (embryo 86-right, 50 mm. crown-rump length, age about 11 weeks).—Loose vascular connective tissue fills the definitely delimited middle ear. The auditory tube extends to the middle ear.

Fig. 3 (embryo 12-left, 135 mm. crown-rump length, age 17 weeks).—Condition much the same as in figure 2. The auditory tube extends deeper into the middle ear, and the connective tissue of the middle ear is looser and extends between the cartilaginous otic capsule and the tympanic plate (*T. P.*) toward the region where the antrum is to form.

Fig. 4 (embryo 45 B-right, 180 mm. crown-rump length, age 21 weeks).—Otic capsule ossifying. The region of the antrum is still made up of cartilage. The connective tissue of the middle ear invading it between the ossifying otic capsule and the tympanic plate is indicated by an arrow.

In these and the following figures, *A.* indicates the antrum; *A. B.*, the antral periosteal bone; *A. T.*, the auditory (eustachian) tube; *A. A. C.*, the antral air cells; *C. A.*, the carotid artery; *C. A. C.*, the carotid air cells; *C. C.*, the carotid canal; *Co.*, the cochlea; *G. G.*, the geniculate ganglion; *E. A. M.*, the external auditory meatus; *I.*, the incus; *I. A. M.*, the internal auditory meatus; *L. A.*, the ampulla of the lateral canal; *L. C.*, the lateral semicircular canal; *M.*, the malleus; *M. A. C.*, the mastoid air cells; *M. E.*, the middle ear; *N. Ph.*, the nasopharynx; *P. C.*, the postcarotid air cells; *S. A.*, the ampulla of the superior canal; *S. A. F.*, the connective tissue of the subarcuate fossa; *S. C.*, the superior semicircular canal; *S. C. A. C.*, the supracochlear air cells; *St.*, the stapes; *T. I.*, the trigeminal impression; *T. N.*, the trigeminal nerve; *T. P.*, the tympanic plate; *T. T. M.*, the tensor tympani muscle, and *V.*, the vestibule.


I.AM.
A.B.
M.E.
A.
T.P.
5
M.E.
A.
A.B.
T.P.
6
A.
M.E.
7
M.E.
A.
8

Figs. 5 to 8.—Sections through the region of the antrum from 4 embryos ranging in age from 22 to 34 weeks. In figure 5 (embryo 85 B-left, 193 mm. crown-rump length, age 22 weeks) the loose connective tissue of the middle ear has extended between the ossifying otic capsule (*A. B.*) and the tympanic plate (*T. P.*) into the region of the antrum. The periosteal bone (*A. B.*) of that region is clearly shown but has not as yet united with the bone of the tympanic plate. The middle ear at this level is filled with connective tissue. Lower in the middle ear the epithelium from the auditory tube has pneumatized about one third of the connective tissue of the middle ear. In figure 6 (embryo 45 A-left, 260 mm. crown-rump length, age 29 weeks) the otic capsule is rather well ossified. The middle ear is largely pneumatized, and the epithelium is seen extending toward the region of the antrum. The connective tissue antrum (*A.*) is rounding out, and the antral bone (*A. B.*) has united around the antrum with the tympanic plate (*T. P.*). Figure 7 (embryo 45 A-right, 260 mm. crown-rump length, age 29 weeks) represents the right ear of the embryo whose left ear is shown in figure 6. The antrum is better developed on the right, and the epithelium of the middle ear has reached the mouth of the antrum. The antral bone has grown laterally, and within it the antrum is considerably larger than that on the left side. In figure 8 (embryo 68-left, 310 mm. crown-rump length, age 34 weeks) the antrum is large, and one half of it is pneumatized. It has expanded through the periosteal bone and is eroding the capsular bone. It is also enlarging laterally, invading the laterally growing tympanic plate and antral bone. This laterally and ventrally growing bone gives rise during childhood to the mastoid process.

of the antrum (*A.*) and is beginning to invade the newly formed, periosteal antral bone (*A. B.*), which expands laterally and fuses with the bone of the tympanic plate around the antrum in fetuses of about 29 weeks (fig. 6). The progressive development of the perichondral bone during childhood gives rise to the mastoid process.

In figure 6, a section of the left ear of a human fetus at the age of 29 weeks, the connective tissue antrum has enlarged within the antral bone, which is just uniting with the tympanic plate. The middle ear is still partly connective tissue, but in it four epithelium-lined air spaces can be seen; this pneumatization, however, has not reached the antrum. In the right ear of the same fetus, shown in figure 7, the process of pneumatization is further advanced, involving most of the middle ear, and has reached the mouth of the antrum. The antrum is considerably larger than that on the left side.

At the age of 34 weeks (fig. 8) the antrum is partly pneumatized and is expanding not only laterally in the direction of the future mastoid but medially into the endochondral bone of the otic capsule. Pneumatization of the antrum must occur rapidly at this stage, for in another fetus, slightly older than 34 weeks (fig. 9), it is well pneumatized.

Mastoid and Antral Air Cells.—In figure 9 the antral bone, which has fused with the tympanic plate, is expanding laterally to form the mastoid process. This process, as is well known, continues to grow until puberty. From the antrum, epithelial buds invade it to form the mastoid air cells (*M. A. C.*, fig. 9), almost as rapidly as the bone is laid down. This mastoid bone, therefore, does not have time to become dense, nor does bone marrow have a chance to form between the bony spicules. In rare instances, when pneumatization for some reason is slowed, bone marrow may form. Mastoid air cells thus begin to develop long before birth. It is true that many of these early formed cells may become incorporated in the future antrum, but pneumatization keeps pace with growth of bone in the mastoid region. While the mastoid protuberance does not become prominent until after birth, yet whatever mastoid bone there is before birth becomes pneumatized almost as soon as it is formed.

Pneumatization also extends from the antrum into the endochondral bone of the capsule to form the antral air cells in the region of the canal. In figure 9 this first evidence of formation of air cells is indicated by the arrow. However the capsular bone has been formed some time before pneumatization begins, so that primitive bone marrow has had occasion to form. Such primitive bone marrow is seen in the bone, to the right of the arrow in figure 9. In the bone ahead of the arrow the primitive bone marrow has lost its cells and has dedifferentiated into a loose mesenchymal connective tissue into which the epithelial

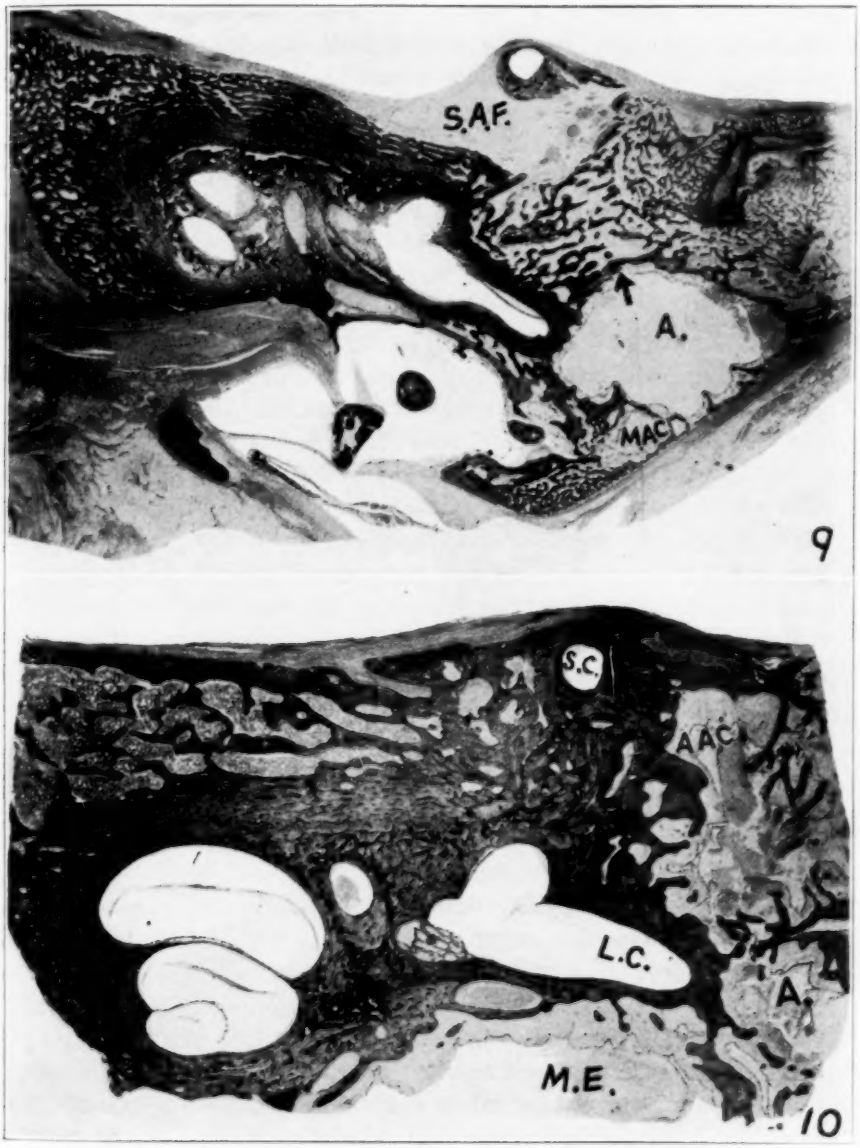


Fig. 9 (embryo 74-left, 315 mm. crown-rump length, age 34 + weeks).—The pneumatized antrum is extending into the capsular bone at the arrow to form antral air cells. It is also extending laterally to form air cells in the region of the primitive mastoid at *M. A. C.*

Fig. 10 (baby 121-left, age 6 months).—Extensive pneumatization from the antrum into the capsular bone lateral to the superior canal to form the antral air cells (*A. A. C.*).

buds may penetrate in the process of pneumatization. In figure 10, a section from an infant at the age of 6 months, the antral air cells (*A. A. C.*) have extensively invaded the capsular bone. A still greater degree of pneumatization by antral air cells is shown in a specimen from a 6 year old child (figs. 11 to 14). Here the antral cells extend far into the otic capsule in the region of the canal. In figure 13 they extend into the arch of the superior semicircular canal and communicate with the supracochlear air cells of the petrous bone. In figure 14 they are seen above the superior semicircular canal.

ORIGIN AND DISTRIBUTION OF PETROUS AIR CELLS

The presence of air cells in the petrous bone has long been known. Regarding their distribution and origin, information is meager. The term "apical air cells" has been generally used for designating petrous cells. Guild [1] pointed out that many of the so-called apical cells do not lie in the apex of the petrous bone and that the groups of air cells should be designated more specifically by location.

While air cells may be found in almost any region of the petrous bone in different persons, yet our observations indicate that they occur most commonly in specific regions. Accordingly in this paper we shall refer to the groups of air cells by names which as nearly as possible describe their locations.

Antral Air Cells.—The antral cells have already been described in connection with the antrum. They are commonly regarded as part of the mastoid cells. However, they lie in the petrous bone lateral to the semicircular canals and thus, from an anatomic point of view, belong to the petrous group of cells. Developmentally they are an outpouching from the antrum into the otic capsule. Clinically, however, they are intimately related to the mastoid cells, because they are continuous with them through the antrum.

Carotid Air Cells.—Next to mastoid and antral air cells, air cells around the carotid canal are most commonly seen. Their extent and distribution vary greatly. We have observed them in four regions: 1. Anterior to the carotid canal and below the auditory (eustachian) tube. This is a subtubal position, and the cells may be called subtubal air cells. 2. Medial and posterior to the carotid canal. Cells in this position might be called postcarotid air cells. They are extensions of the subtubal cells. 3. Between the auditory (eustachian) tube and the carotid canal. The cells may be extensions of the subtubal cells, or they may open into the middle ear or eustachian tube separately. They may be called precarotid air cells. 4. Lateral to the carotid canal, between the mouth of the eustachian tube or the tensor tympani muscle and the

apex of the cochlea. This is a precochlear position, and the cells may be called precochlear air cells.

Carotid air cells develop much the same as do those in the mastoid. There is at first a loosening and widening of the connective tissue spaces in the newly formed bone. These loose connective tissue spaces usually appear soon after the bone is laid down. Epithelial buds from the middle ear or in some cases from the auditory (eustachian) tube invade them to form air cells. They make their appearance in fetuses of 210 to 280 mm., or at the age of 24 to 31 weeks, and the epithelial invasion occurs soon thereafter. The earliest epithelial inbudding was observed in a fetus of 246 mm., or at the age of 27½ weeks. The process of expansion of connective tissue and epithelium continues into childhood and possibly later. Apparently it follows closely the deposition of bone. Marked differences occur in different persons. In some the pneumatization is extensive and in others it is arrested early, either extensive or limited formation of air cells thus resulting.

1. The subtubal air cells are more extensive and more constant than any of the other carotid groups. They are present in 88 per cent of the ears studied from fetuses over 27 weeks old and in 90 per cent of those from infants up to 6 years. Of the ears which contain them only a small percentage have extensive pneumatization; the majority have only slight formation of air cells. Different stages of formation of subtubal air cells are shown in figures 15 to 18. In figure 15 one of the early stages is shown. Here only the pockets of loose connective tissue (*C. A. C.*) are formed in the bone anterior to the carotid canal. Figure 16 is a section from a fetus at term. The large connective tissue pockets are partly pneumatized. The bone around them is compact, so that extension of the air cells is probably limited. In an infant of 6 months (fig. 17) there is an extensive loosening of connective tissue, although the epithelial invasion has not progressed far. The loose connective tissue in this specimen has extended to the medial side of the carotid artery and is invading the bone posterior to it, giving rise to the second group of carotid air cells. In a 10 month old baby, from whom the section shown in figure 18 was taken, the subtubal air cells are extensive and well developed. However, they do not extend to the posterior side of the carotid canal.

Subtubal cells, as a rule, open into the middle ear, but occasionally some of them open into the auditory tube. In figure 18, most of the air cells open into the auditory (eustachian) tube, and only those open into the middle ear which are nearest it. A reconstruction of these air cells with their connection to the auditory tube is shown in figures 25 and 26.

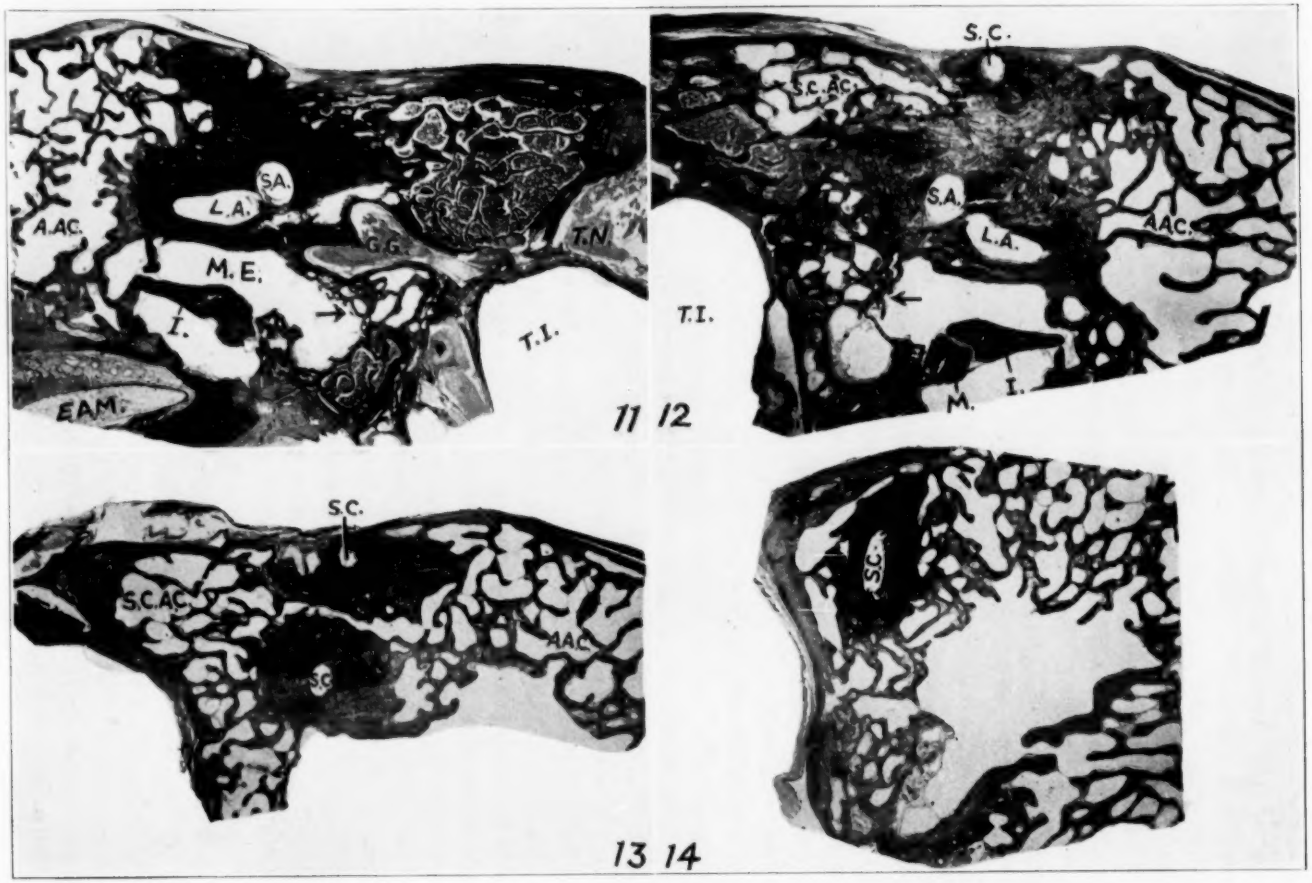
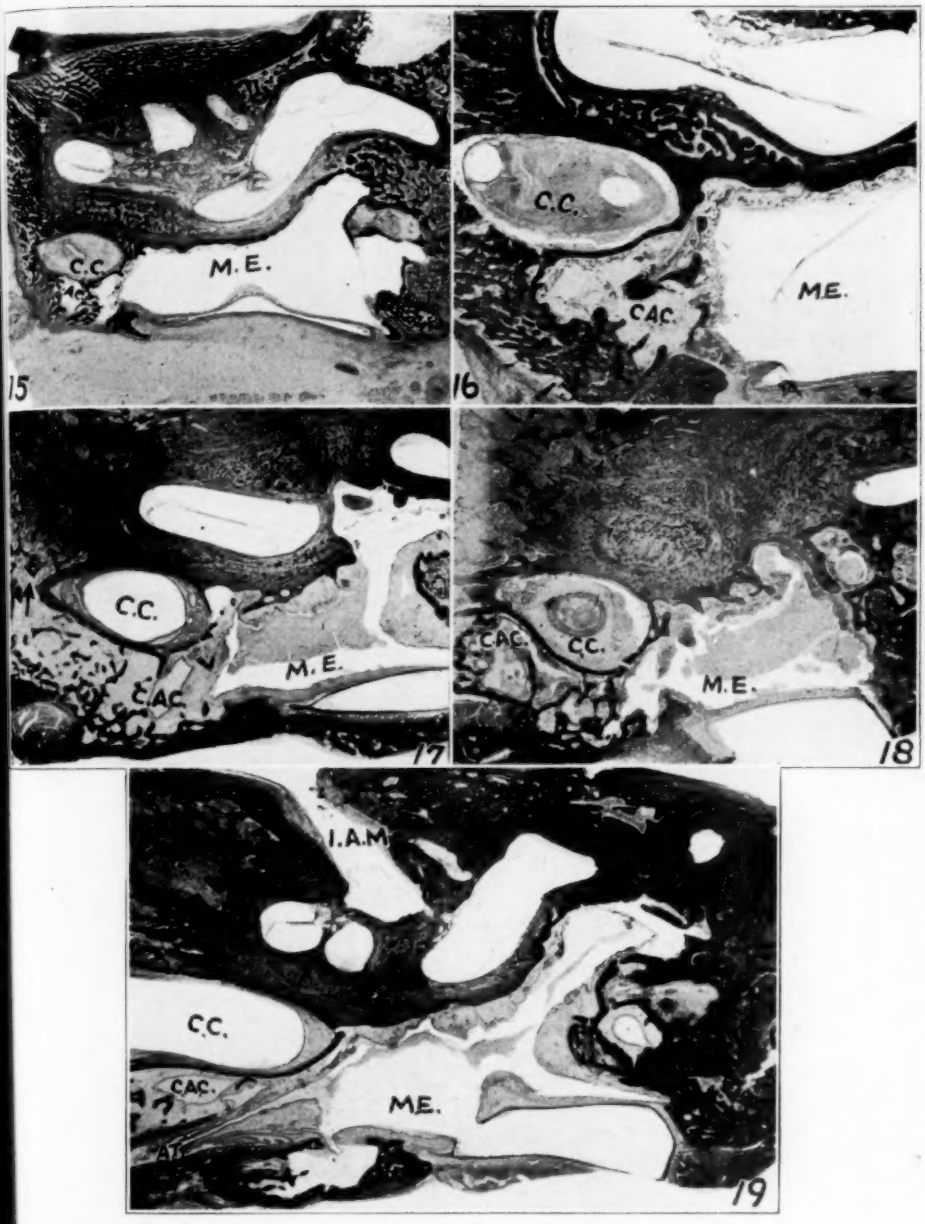


Fig. 11 (baby 81-right, age 6 years).—Two areas of pneumatization, the one an extension from the antrum into the capsular bone lateral to the superior semicircular canal (*A. A. C.*) and the other from the middle ear into the otic capsule anterior to and above the geniculate ganglion. The air cells of the latter are shown ahead of the arrow and are the supracochlear air cells.

Figs. 12 to 14 (baby 81 left, age 6 years).—Sections from the left ear, showing the upward extension of the two areas



Figs. 15 to 19.—Successive stages in the formation of air cells around the carotid canal below the auditory tube (figs. 15-18) and a few air cells between the auditory tube and the carotid canal (fig. 19). Figure 15 (embryo 116 A-left, 280 mm. crown-rump length, age 31 weeks) shows the invasion of loose connective tissue from the middle ear into the newly forming bone anterior to the carotid canal and below the auditory tube. The invading structures are the developing subtubal air cells (*C. A. C.*). In figure 16 (baby 95-left, at term) the area shown in figure 15 is partly pneumatized (*C. A. C.*). In figure 17 (baby 98-left, age 6 months) extensive loose connective tissue invades the bone posterior and medial to the carotid canal, showing some pneumatization. The arrow points to the early pneumatization of the postcarotid air cells. Figure 18 (baby 97-left, age 10 months) shows extensive pneumatization anterior and medial to the carotid canal. Figure 19 (baby 98-left, age 6 months) shows pneumatization between the auditory tube and the carotid canal. The pneumatic spaces are the precarotid air cells.

2. The postcarotid air cells lie medial and posterior to the carotid canal and medial to the cochlea. Anson, Wilson and Gaardsmoe[3] designated them as group D. We have seen them clearly in only 2 specimens of our series. In 1, from a 6 month old infant (fig. 17), the loose connective tissue from the subtubal region is invading the postcarotid region, indicated by an arrow. This process occurs in older bone, where bone marrow is already present. The bone marrow loses its cells, and the tissue becomes loose. This process is like the process of formation of antral air cells extending into the otic capsule in the canal region. The second specimen is from a premature infant which lived two days. It measured 345 mm., which corresponds to a fetal age of 37 plus weeks. A section through its petrous bone is shown in figure 24. The postcarotid air cells (*P. C.*) are fairly well formed and are partly pneumatized. Their medial extent is not known because the extreme apex of the petrous bone was cut away. A row of air cells extending from them along the anterior side of the carotid canal communicates with the subtubal air cells, which in turn open to the middle ear.

3. The precarotid air cells are not constant. They lie between the auditory tube and the carotid canal. Sometimes they open into, and must be considered an upward extension of, the subtubal cells. At other times they open separately, either into the mouth of the auditory tube, as is shown in figure 19 (*C. A. C.*), or into the middle ear between the tube and the carotid canal. The precarotid and the subtubal group correspond to group C of Anson, Wilson and Gaardsmoe.[3]

4. The precochlear group of air cells lie lateral to the carotid canal between the upper part of the auditory tube and the apex of the cochlea, as shown at *X* in figures 20 and 21, and extend upward between the tensor tympani muscle and the apex of the cochlea, as shown at *X* in figure 22. This is a fairly constant group of air cells but as a rule is not extensively developed. Usually it consists of a mere pocket from the middle ear, although sometimes there is an extensive excavation of the bone over the apex of the cochlea (*X*, fig. 22). It corresponds to group A of Anson, Wilson and Gaardsmoe.[3] Their group B, which is an upward extension of group A, was not observed by us.

Supracochlear Air Cells.—The supracochlear air cells lie above the cochlea, anterior to and above the geniculate ganglion. They may extend into the petrosal ridge and from there, laterally, toward the arch of the superior semicircular canal. They arise from the middle ear above the hamulus of the tensor tympani muscle, at about the level of the incus-stapes joint. They are variably developed. Sometimes they appear as upward extensions of the precochlear group and extend hardly up to the level of the geniculate ganglion. At other times they

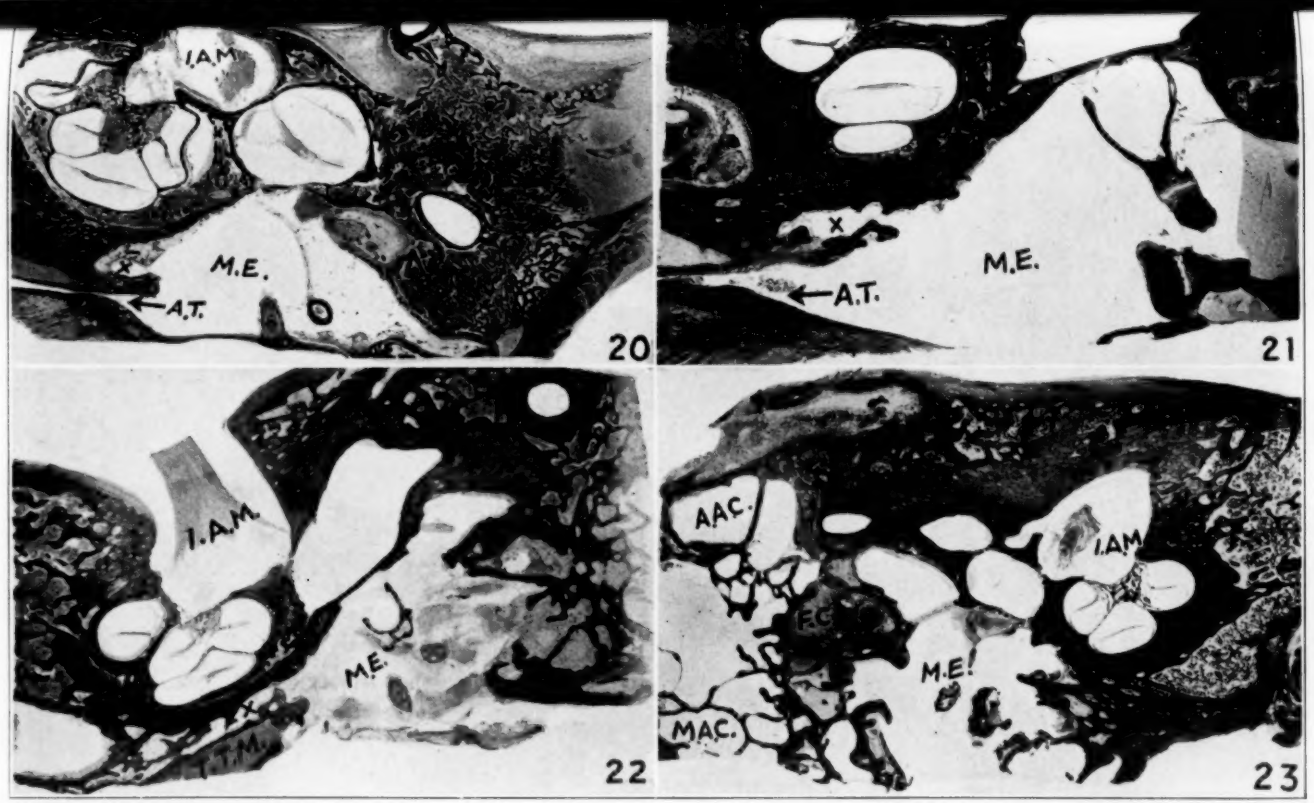


Fig. 20 (embryo 113-left, 235 mm. crown-rump length, age 26 weeks).—Small area of pneumatization between the auditory tube and the tip of the cochlea at *X*. This is an early stage in the formation of the precarotid air cells.

Fig. 21 (baby 123-left, at term).—More advanced condition of the area of pneumatization (*X*) shown in figure 20. In figure 22 pneumatization is seen extending upward between the tensor tympanic muscle and the cochlea.

Fig. 22 (baby 120-left, age 7 months 13 days).—Precochlear air cells, at *X*, between the tensor tympani muscle and the cochlea.

Fig. 23 (baby 88-right, age 2½ years).—Marked pneumatization around the facial canal. Some of the air cells are derived from the antrum, and some extend directly from the middle ear to the mastoid air cells.

are distinct from the precochlear group. As a distinct group they were found in a relatively small percentage of our petrous bones.

A well defined example may be seen in our serial sections of the temporal bones of a 6 year old child, pictured in figures 11 to 14. Figure 11 represents a section through the right petrous bone and shows the air cells anterior to the geniculate ganglion, emerging from the middle ear at a point indicated by an arrow. Figure 12 represents a similar section from the left petrous bone, cut just above the geniculate ganglion. The arrow points toward a group of cells extending into the bone in a posterior direction, toward a larger group of air cells (*S. C. A. C.*) with which they are shown to be continuous in figure 3, representing a section at a higher level in which these supracochlear cells (*S. C. A. C.*) extend laterally and communicate with the antral cells (*A. A. C.*) through the arch of the superior semicircular canal. Some of them even extend over the superior semicircular canal, as is shown in figure 14, which represents a section of the same petrous bone at a higher level. The cells of the supracochlear group which lie high in the petrous bone have been referred to as attic cells by other authors.

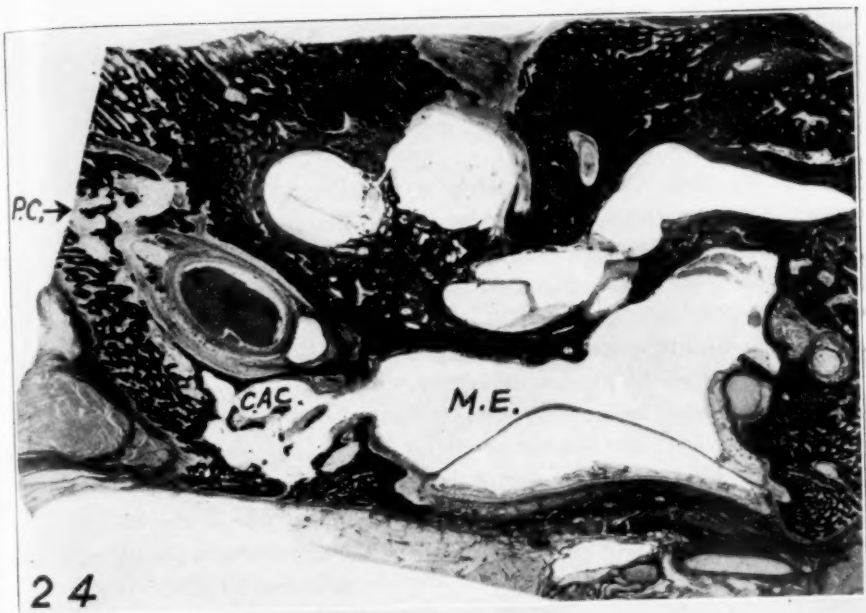
In the other petrous bones in which the supracochlear group of air cells was seen, they occupied only a small area in the region of the geniculate ganglion. It may be that they develop and expand later, since only the maturest specimen of our series, that from a 6 year old child, showed them extending far into the periotic capsule. It is of significance that they expand like the antral and postcarotid cells into older capsular bone, whereas most of the other air cells form in periosteal bone almost as soon as it is laid down.

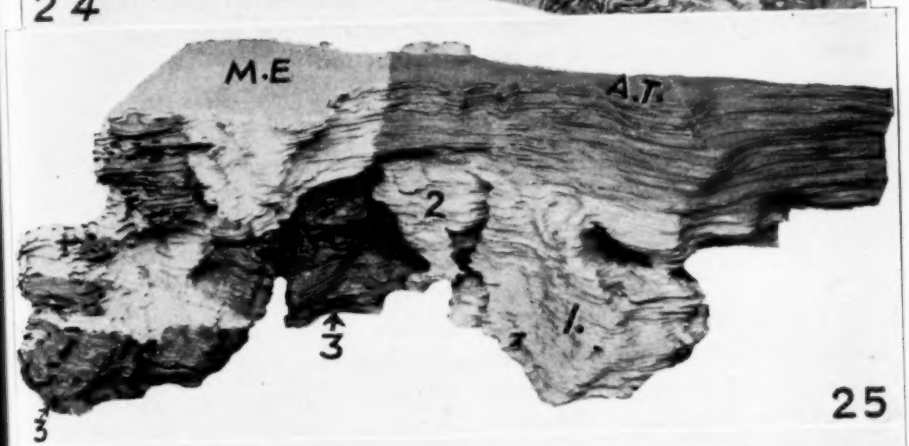
Apical Air Cells.—The term "apical air cells" is used extensively in the recent literature. In our collection we have not seen air cells in the apical portion of the petrous bone forming a separate group. The postcarotid air cells extend far into the apex and might be considered apical in position. Anson, Wilson and Gaardsmoe [3] showed some of the carotid air cells in a 4½ year old child extending far into the apical portion of the petrous bone, but they stated that they did not consider them a separate group of air cells.

OUTLET OF AIR CELLS

The outlets of the various groups of air cells are important in determining the portals of entry of infections. As observed in our collection, they are as follows:

The mastoid air cells open as a rule into the antrum. In a few specimens observed they open low into the middle ear, directly anterior to the facial canal. An example of this is shown in figure 23, representing a specimen in which the wall between the middle ear and the mastoid cells is highly pneumatized.





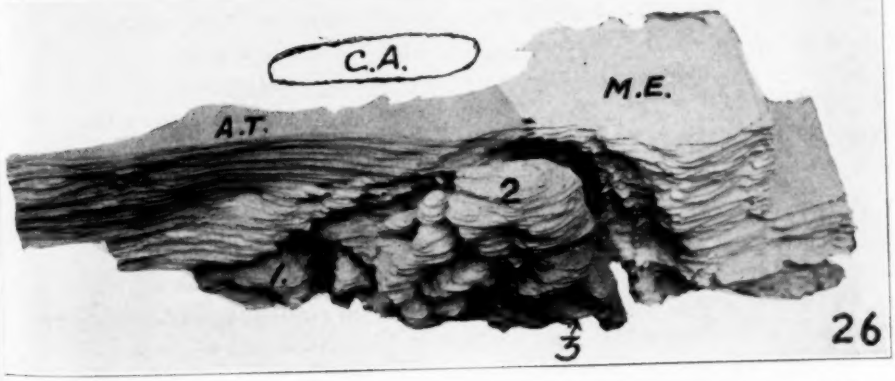


Fig. 24.—Section through the left petrous bone of a 2 day old premature child (size 345 mm., corresponding to a fetal age of 37 weeks). The subtubal air cells are shown at *C. A. C.* and the postcarotid air cells at *P. C.* A few cells are seen extending between these two groups.

Figs. 25 and 26 (baby 97, age 10 months).—Reconstruction of part of the middle ear and part of the auditory (eustachian) tube, with groups of air cells, *1* and *2*, which open into the auditory tube, and *3*, which opens into the middle ear. The position of the carotid artery is indicated at *C. A.* Figure 25 is a posterior slightly superior, and figure 26 an anterior slightly superior, view.

The antral air cells open into the antrum.

Of the carotid air cells, the subtubal cells open into the middle ear, below the eustachian tube. In a small percentage of bones some of them open directly into the auditory (eustachian) tube. In figure 18 the group of cells marked *C. A. C.* open directly into the auditory tube. A reconstruction of these subtubal air cells is shown in figures 25 and 26. Two groups of these cells, marked *1* and *2,* open into the auditory tube, and group *3* opens into the middle ear. The postcarotid cells are, as has been seen, extensions of the subtubal cells and therefore open with them into the middle ear or on rare occasions into the eustachian tube. The precarotid cells open either into the mouth of the eustachian tube, as seen in figure 19, directly into the middle ear between the mouth of the tube and the carotid canal or into the subtubal air cells. The precochlear cells open into the middle ear between the upper part of the mouth of the eustachian tube and the tip of the cochlea.

The supracochlear air cells open into the middle ear in the region medial to and above the hamulus of the tensor tympani muscle or a little below the level of the geniculate ganglion. The arrow in figures 11 and 12 point to this region. Sometimes they seem to be continuous with the precochlear group.

INTERRELATION OF GROUPS OF AIR CELLS

That there is an interrelation between certain groups of air cells is well known. For example, the antral cells are continuous with the mastoid cells through the antrum. Whether there is any continuity between other groups of petrous air cells or between them and the mastoid cells is still a question. We have observed communication of air cells in several specimens. The carotid group are more or less continuous. Thus, the postcarotid cells are usually continuous with the subtubal air cells. The precarotid cells may be continuous with either the subtubal or the precochlear group. There is evidence also that the supracochlear group may be continuous in some specimens with the precochlear. We are not certain of this connection, and reconstructions will be necessary.

The supracochlear group, however, is sometimes connected with the antral air cells through the arch of, and over, the superior semicircular canal. This connection may be seen in figures 13 and 14, which represent the only specimen in which we have noted it.

RELATION OF AIR CELLS TO BONE MARROW

From our study we must conclude that there is no direct relation between air cells and bone marrow. Air cells do not invade bone marrow directly. When air cells extend into diploic bone, the cells of

the bone marrow disappear, and the connective tissue becomes less dense than prior to the invasion of the air cells. Most air cells are formed in young growing bone before bone marrow is formed. Only the antral, the postcarotid and part of the supracochlear air cells invade bone with bone marrow, and in these bones the marrow recedes before the air cells enter. When the formation of air cells is complete the cells are separated from the bone marrow either by plates of bone or by dense connective septums.

In this connection it is significant to note that certain pathologic air spaces may occur directly in bone marrow. We have seen several specimens in which numerous large air spaces occurred in the bone marrow of the apical portion of the petrous bone. At first we thought them to be true air cells, but when the sections were studied in series they proved to be closed spaces. They did *not* communicate with the middle ear or with the true air spaces. Furthermore, they were not lined by true epithelium but were surrounded by compressed bone marrow. In 1 specimen they contained fungus-like growths and spore-like bodies. Apparently a gas-forming growth had produced these large spaces in the bone marrow. Whether this condition occurred during life or post mortem has not been determined. We are mentioning it here because the gross error of interpreting such spaces as true air cells is possible.

PERCENTAGE OF PNEUMATIZATION OF PETROUS BONE

A review of the literature shows that there is no agreement as to the percentage of pneumatization. The figures vary greatly. In presenting those based on our observation we wish to state that percentages cannot be accurately stated because certain regions of the petrous bones where air cells may occur were cut away prior to sectioning and some sections were badly torn in regions where air cells occur. Thus the region where the subtubal cells occur was removed in a number of specimens, so that the total from which to derive percentages was reduced. The accompanying table gives as accurately as possible the percentage of occurrence of the groups of air cells in human infants ranging in age from term to 6 years. Under the total percentages all specimens were included, from those with a mere indication of formation of air cells to those in which the air cells were extensively developed. The distinction between "Large" and "Small" is an arbitrary one, but only the specimens with extensively developed cells are included under "Large." Therefore, in those included under "Small" the degree of development of air cells varied from a mere indication to a moderate group of cells.

The postcarotid cells are not listed in the table because we have seen them distinctly in only a few specimens. Since they appear to be an

extension of large subtubal cells, it is probable that they occur only when the subtubal cells are extensively developed. It is also difficult at times to determine in which group certain cells are to be placed.

The percentages of the groups of air cells in fetuses are not given because the age at which they develop is subject to considerable individual variation. In some fetuses certain air cells may begin to form at the age of 24 weeks. The same air cells may not appear in other fetuses until the age of 32 weeks. If one considers only fetuses of 8 months or older, the percentages are slightly lower but approximate nearly those listed for infants in the table. This indicates that most of the groups of air cells have started to form in fetuses of 8 months, although a few may appear later. It should be remembered, however, that they continue their growth and expansion into the growing bone possibly throughout childhood.

Number and Percentage of Groups of Air Cells in Ears of Infants Ranging in Age from Term to Six Years

	Subtubal Air Cells		Precarotid Air Cells		Precochlear Air Cells		Supracochlear Air Cells	
	No.	%	No.	%	No.	%	No.	%
Large*	9	30	..	..	9	25	4	11+
Small†	18	60	12	40	23	64	16	44+
None	3	10	18	60	4	11	16	44+
Total number	30		30		36		36	
Total % of air cells		90%		40%		89%		56%

* Extensive pneumatization.
† From air cells just indicated to groups which are not extensive.

SUMMARY

In an 8 week fetus the auditory (eustachian) tube extends to the middle ear. The connective tissue of the middle ear then becomes loose, and the epithelium from the auditory tube invades it. This pneumatization of the middle ear is slow and is not completed until late in fetal life.

The region of the antrum is first indicated in the twenty-second week of fetal life, when the loosening connective tissue from the middle ear extends laterally between the tympanic plate and the otic capsule in the region of the canal. The periosteal bone of the capsule then grows around this loose connective tissue and unites with the tympanic plate in the twenty-ninth week of fetal life. Soon after this the epithelium from the middle ear invades the loose connective tissue of the antrum. The antrum is well pneumatized by the thirty-fifth week.

With the expansion of the antral bone and the tympanic plate to give rise to the region of the mastoid, the epithelium from the antrum invades the newly forming bone to form the mastoid cells. Some of these early cells later become incorporated in the enlarging antrum.

Air cells from the antrum also invade the bone lateral to the semicircular canals to form the antral cells.

As the petrous bone develops, other air cells are formed in it. One large group, formed in the bone around the carotid canal, can be subdivided into four smaller groups, the subtubal, the postcarotid, the precarotid and the precochlear. Another group, the supracochlear, lies anterior to and above the region of the geniculate ganglion.

No true apical air cells were seen other than those of the postcarotid group, which extend far into the apical part of the petrous bone.

Mastoid cells as a rule open into the antrum, but occasionally they open into the middle ear directly. Antral air cells open into the antrum. The carotid group of cells open into the middle ear, but the subtubal and precarotid cells may also open into the auditory (eustachian) tube. The supracochlear cells may open into the middle ear or be continuous with the precochlear group.

Intercommunication between groups of air cells does occur. It is common between the subgroups of the carotid air cells. It was seen in 1 specimen also between the antral cells and the supracochlear cells.

Air cells do not extend into true bone marrow.

Formation of air cells occurs as a direct extension of epithelial buds into newly forming bone, or the process may invade older bone with bone marrow. In the latter case, bone marrow cells disappear, and loose, acellular connective tissue forms prior to the invasion of the epithelium.

Large spaces in bone marrow resembling air cells are sometimes seen in histologic sections, but they are not true air cells. They are due either to some pathologic change or to postmortem formation of gas.

The frequency of pneumatization of the petrous bone is shown in the table.

HYPNOSIS

A POTENT THERAPY FOR CERTAIN DISORDERS OF VOICE AND SPEECH

JOHN J. LEVBARG, M.D.
NEW YORK

At the present time there is an increasing emphasis placed on the correction of voice and speech defects by colleges, universities and public school systems; there is also an awakening interest in the subject on the part of numerous physicians, medical schools and hospitals. Every one appreciates speech as a medium for social communication. All men from the lowliest laborer to the most dignified professional man admire and respect a person possessing the ability to express himself properly. In fact, it is more often remarked that the speech of a physician is more tonic than his medicine. As a writer appropriately stated, "the human voice is the most delicately tuned musical instrument that God has created. It is capable of cultivation beyond the dreams of those who have given it no thought. It may be made to express every emotion in the gamut of human sensation from abject misery to boundless ecstasy."

As a rule disorders of the voice and speech mechanism are functional in character and psychic in origin. It is up to the physician to seek the proper causes producing such a disturbance before he can look for a cure. It is my purpose in this short article to present a method of treatment suggested in part from experiments in the psychiatric department of the New York Hospital, when I was associated with Dr. Blanton in work on the correction of speech defects. Dr. Blanton, who is a psychiatrist, used mainly psychoanalytic treatment, but that method would take a long period, and in many instances the patients would not continue treatment. Dr. Blanton in referring patients to me would often remark "that I should use psychotherapy in the form of suggestion." But possessing mainly a musical and otolaryngologic knowledge, I held myself aloof for three years before I undertook to apply hypnosis to the correction of voice and speech defects. The present report is the outcome of experience gathered in the course of unwearying research covering a period of two years with patients at the speech clinic of the Harlem Eye and Ear Hospital and from my own private practice.

Presented at the monthly staff meeting of the Voice and Speech Department of the Harlem Eye and Ear Hospital, with demonstrations of recordings of voice and speech defects and presentation of cases.

In conjunction with my reeducational work, i. e., exercises of the lips, jaw, tongue and throat muscles, plus auditory stimulation through vocalization and ear training, I am increasingly applying hypnosis. This procedure, as stated, has been in use for about two years, and the results encourage the hope that it will interest others who are doing work in the correction of voice and speech defects. Since last year I have also applied it to my otolaryngologic specialty in the electrocoagulation of tonsils, paracentesis of ear drums and the diminishing of concha bones and as an agent for calming the patient, especially singers and those who use speech a great deal.

Hypnosis is one of the most practical sciences today, and it confers advantages which could not be obtained by any other medium. It overawes every one who witnesses its marvelous manifestations. It is especially practical for dysphemias, abnormal self consciousness and pathologic diffidence, and it also aids to destroy destructive habits. It is greatly instrumental in instilling healthy mental attitudes and motivates toward better coordination. As a physician I do not use it for frivolous purposes but expressly for curative and scientific treatment.

Hypnosis is merely a form of persuasive suggestion. Its principle and method of procedure are simple. However, it rests with the physician as to when to use it, as there are no two cases alike. The results obtained with this form of therapy, which has been used in about 75 cases of speech disorder representing various dysfunctions, in a period of two years have been remarkable. The period of correction has been cut from six months to practically three and in some cases even less. Patients are more confident, possess greater assurance, are more dynamic and are able to speak and express themselves freely and more successfully with a forwardness which was lacking previously. They develop a greater emotional stability and become better adjusted socially.

It has been my good fortune to have in this group of patients studied a number of stammerers and stutterers with marked anxiety neurosis, hysterical aphonias, choreic speech, spastic speech and dysphonia pubetica; the majority of such persons suffer from variable symptoms, such as nervousness, shyness, diffidence, embarrassment and fear. In some persons these symptoms are prominent; in others they are mild. At the beginning I selected 20 patients from the voice and speech clinic whom I thought might respond to hypersuggestion, and arrangements were made for subsequent observation at intervals of one week apart. In my own private work some patients were given two and others three sessions per week. Improvement was immediately manifested in their mental and physical makeup. Of the first 20 persons, 8 with fewer of the classic symptoms responded in two sessions, but they are still under observation, receiving relaxation and vocal exercises for further flexibility of their speech. For 5 patients hypersuggestion was used

exclusively, with marked improvement. Posthypnotic suggestion was given to assist the patients to meet the problems of life. They were encouraged to exercise self discipline and self confidence, to improve their thoughts with cheerful and pleasant subjects and not to be too introvertish. Mental and physical hygiene was not overlooked.

As heretofore mentioned, the most common disorders encountered were stammering and stuttering. This I found to be true both in the voice and speech clinic and in my own private practice. The terms stammering and stuttering are used interchangeably by some speech pathologists, but to distinguish them from the layman's point of view, stammering is a condition in which one cannot start, whereas stuttering is a repetition of sound—in other words, one gets started but cannot stop. With either disorder the patient is neurotically inclined, anxious and fearful, and the speech mechanism is momentarily paralyzed. This paralysis may be due to a spasm of either the throat, lips, glottis or respiratory muscles. The patient cannot think clearly while speaking to another, is always on the watch for words he thinks he cannot say, speaks rapidly, feels embarrassed and self conscious and cannot adjust himself adequately. In a great majority this effect is accompanied by complete somatic contractions, tensions, spasms, twitchings, clenching of the jaw or blinking of the eyes. In my opinion the defect is not in making the beginning sounds but in relaxing the muscles for one moment in time to produce the next. Rarely is there blocking or repetition of a consonant at the end of a syllable. Other speech and voice defects in this group were dysphonia pubetica, i. e., a high pitched falsetto voice, and hysterical aphonia, in which there was either a total loss of the voice or a condition producing merely breaks in the rhythm. Spastic and choreic speech were also considered for experimentation. Choreic speech is largely due to a motor disability with a great deal of incoordinated movement of the speech organs.

One of the greatest drawbacks for complete success with all these patients is the consciousness of introversion. Such persons admit an inadequacy and manifest a negative condition which is vital to progress and success. I have found that suggestion in the waking state is not readily accepted, but when it is remembered that hypnosis is the art of mentally controlling the thoughts and actions of a patient, one can realize the great value it possesses to those who employ it in the practice of correcting speech disorders.

Hypersuggestion can be readily used, as most of these persons suffer from various forms of nervousness which have no actual cause for their existence. Their morbid self consciousness severely interferes with their success in life. They are especially excessively nervous in the presence of strangers. Some have cold perspiration over the entire body, the heart beats rapidly, the voice falters, respiration fails them,

and they are a picture of confusion. Hypnosis is greatly instrumental in strengthening the morale of these victims and in helping them to meet the exigencies of everyday life. It is absolutely necessary to ingrain in the mind the feeling of adequacy and equality, and all sense of inferiority in the presence of strangers and acquaintances must be removed.

The importance of instilling a new habit in the mind of such a person is essential. Every one knows that every move or voluntary action is controlled by one's thoughts, and this continued nervous force makes definite imprints on the brain tissue. When this nervous energy is repeated, lasting impressions are made, and thereafter the action practically becomes almost involuntary. This agrees with Pavlov's experiments on conditioned reflexes in dogs. By repeatedly emphasizing a certain action in the same manner, a lasting impression is established in the dog.

Under hypnosis a patient with a speech disorder readily becomes passive and receptive, and his psychic resistance is easily overcome. When the speech pathologist suggests thoughts of recovery, thus making positive ideas penetrate the mind, the patient readily believes symptoms no longer exist or that they will disappear. One is instilling faith and confidence in the patient and is reenforcing constructively the physical, mental, moral, social and economic status of such a person. In other words, the brain accepts and transforms suggestions into a reality. This belief becomes fixed and is not easily diverted by negative waking suggestions.

The following cases, selected from the group of various forms of speech disorders, demonstrate the response to the practical application of hypnosis.

REPORT OF CASES

CASE 1.—Samuel S., a youth aged 18, came to the clinic from April 17 until Oct. 9, 1937. His condition was diagnosed as dysphemia clonica, a substitution of sound and oral inactivity. He went through elementary school in ungraded classes. While his mental level as determined by the Stanford-Binet test was low, this was not believed to be an entirely valid measure of his true mental ability. His shyness, poor speech and poor language background have all probably contributed to make his score low. At an examination with tests involving mechanical ability he did much better, being only somewhat below average. His speech defect was a serious handicap to eventual vocational placement. At the second session he was directed to lie on the couch and to gaze fixedly at a chrome-plated pencil having a round globe tip. He was told to think of sleep and not to resist the physician. In less than three minutes he entered the cataleptic stage, and then posthypnotic suggestions were given. He was seen once a week, and within a month marked improvement in speech was noted. Before he stopped coming to the clinic he obtained a position as a clerk in a grocery store and was getting along splendidly.

CASE 2.—W. W., a shipping clerk aged 24, a stammerer, was first seen on Aug. 1, 1938, at my office. He complained of fear of speaking before strangers. The fear caused a severe dull headache, which was constant and unbearable. Under hypnosis the headache was relieved immediately. The patient received three treatments per week, and within a month correction in speech was evident. At the time of writing the patient is enthusiastic, can speak before strangers and is also able to read before a group. He was going to study for the ministry, but he felt his speech defect to be a serious handicap. He now has renewed ambition to carry out his original intention, and he expects to take up his studies this summer.

CASE 3.—Miss S. F., aged 41, a music teacher, came to my office on Sept. 29, 1938. Owing to emotional shock, her voice would break, and she was unable to sustain a phrase but for a few seconds. Her speech would block, crack and become jerky, and sometimes it was entirely disturbed. The patient is hypertense and easily upset. Weekly sessions under hypnosis inspired confidence and better control of the voice. She is still under my care.

CASE 4.—H. M., aged 16, came to the voice and speech clinic on Jan. 16, 1937, with a high pitched falsetto voice. He was nervous, shy and diffident. Physical examination gave negative results. He had had a deviated septum corrected for sinus trouble. Under waking suggestion the voice dropped one octave at the first session, and at the third session the voice was fuller, vibrant and a man's voice. Under hypnosis nervousness improved. In this case visits to the clinic were irregular, and in October 1937 he was discharged as cured.

CASE 5.—S. E., aged 16, a high school girl, had spastic tonic aphemia. She had undergone a severe shock at the age of 18 months. There was marked blocking of speech, which probably dated back to the period of shock. The girl was brought to the clinic by a woman other than her mother. There was an air of mystery about the patient's mother and the exact nature of the shock. When questioned about it, the patient's defective speech became more marked. She had had a tonsillectomy and wore a brace for oral malocclusion. Other physical characteristics were normal. Her attitude at the clinic was friendly yet reserved. She started coming to the clinic on Feb. 26, 1938, and makes fairly periodic visits. Under hypnosis, rapport was easily established. At the time of writing the girl is still under my supervision, and her blocking is becoming less frequent. Her entire personality seems to be changed, and she reports that her work in school is improving and that she is becoming better adjusted to her environment.

CASE 6.—E. R., aged 18, came to the clinic in April 1938. She was referred by an orthopedist of the Hospital for Joint Diseases for her spastic speech. When she first came to the clinic her posture, motivation and speech were extremely spastic and awkward. She has been very persistent in her visits to the clinic, and at the time of writing her physical characteristics are almost normal. Her speech has greatly improved, her gait is about normal and she has better control over her muscles. Spasticity is greatly abolished. The girl cooperates well in psychotherapy and under hypnosis has established an almost normal pattern of speech. She is still under my care.

SUMMARY

Hypnosis is a potent agent in psychotherapy.

It can be used for the correction of certain disorders of voice with positive results.

A speech pathologist should be a physician.

Such a physician should have training in psychiatry.

He should possess a musical knowledge and be a competent otolaryngologist. As James S. Greene [1] stated, such a physician should be a musically and psychiatrically trained laryngologist and one who will be able to institute "a composite therapy which correlates the psychic and somatic states into an integrated whole, so that the action on the personality is of such a nature that the patient achieves a normal standard of voice and speech."

55 West Forty-Second Street.

1. Greene, J. S.: Dysphemia and Dysphonia: Cardinal Features of Three Types of the Functional Syndrome; Stuttering, Aphonia and Falsetto (Male), Arch. Otolaryng. **26**:74 (July) 1937.

THE EUSTACHIAN TUBE

ABNORMAL PATENCY AND NORMAL PHYSIOLOGIC STATE

H. B. PERLMAN, M.D.
CHICAGO

Johannes Müller,[1] writing about the ear in his monumental handbook of physiology, stated that he could produce a snapping sound in his ears associated with contraction of the palatine muscles and elevation of the soft palate. He said, "If immediately afterwards I emit a humming vocal tone while my mouth is either closed or only slightly open, this tone has an extraordinary resonance." He could not explain this phenomenon but believed that it had something to do with the contraction of the tensor tympani muscle; while he had given much thought to the function of the eustachian tube, he did not correlate the phenomenon with its opening. He believed that it is continuously open. It was Toynbee,[2] in 1853, who concluded from experiments on himself and from the tendency to swallow while descending in a diving bell that the eustachian tube is normally closed and opens only during the act of swallowing. He found that by swallowing with the nose closed (Toynbee's maneuver) a sensation of fulness, or distention, is produced in the ears, which remains on removing the hand from the nose. He concluded that this is due to the forceful entrance of air into the middle ear and that the resultant increase in pressure is maintained by the closed state of the eustachian tube. That this sense of pressure is relieved by swallowing with the nose open indicated to him that the tube opens only during the act of swallowing. It was some time later (1864) that Politzer[3] showed by his experiments with a manometer sealed in the external auditory canal that Toynbee's maneuver results in a negative pressure, or exhaustion of air, in the middle ear. It was noted also that Toynbee's maneuver is usually associated with a distinct movement of the drum. The contention of Toynbee that the tube is

From the Department of Surgery, Division of Otolaryngology, the University of Chicago.

References to periodicals in footnotes are to the page containing the material cited, unless inclusive.

1. Müller, J.: Elements of Physiology, ed. 2, translated by W. Baly, London, Taylor & Walton, 1842, vol. 2, p. 1264.

2. Toynbee, J.: Proc. Roy. Soc. London **6**:286, 1853.

3. Politzer, A.: Text-Book of the Diseases of the Ear, ed. 3, translated by O. Dodd, Philadelphia, Lea Brothers & Co., 1894, p. 63.

normally closed was not readily accepted, especially by otologists on the Continent—not even after Jago,[4] who worked with Toynbee at the Cornwall Infirmary, described in great detail observations on himself of symptoms which he correctly interpreted as due to a continuously open eustachian tube. Jago described and interpreted his observations —last published in 1867, after he had carefully studied his symptoms for over fourteen years and had also studied those of 2 of his patients— so clearly as not to be excelled by any subsequent writer and to establish permanently the clinical entity of the continuously open eustachian tube. He believed, however, that the phenomenon is exceedingly rare—as did Hinton, who wrote about it in Toynbee's textbook on otology.[5] There appeared in the Continental literature at about that time a great deal of material on the function of the eustachian tube, with controversies pertaining to whether the tube is normally open and to the interpretation of autophony. In 1865 Politzer[6] referred to J. Müller's experiments and stated that the phenomenon Müller described, as quoted, was due to opening of the eustachian tube. In 1869 Politzer[7] reported that opening of the eustachian tube during the act of swallowing is shown further by the increase in the loudness of a tuning fork held at the nose. Gruber,[8] Rudinger,[9] Lucae,[10] Weber[11] and Brunner[12] were prominent among the observers entering into a controversy with Toynbee and Jago on the function of the eustachian tube. Brunner in 1870 described in great detail a patient who appears to have had every finding that Jago reported as due to a continuously open eustachian tube but came to the conclusion that he was dealing with "catarrh" of the tube. He was misled because the patient's symptoms were relieved temporarily by inflation of the eustachian tube. Previously, in 1868, Gruber had come to that conclusion in a paper entitled "Tympanophonie und Autophonie." As late as 1883 Brunner[13] was only partially won over to the idea that autophony (a state in which one's own voice and respiratory sounds are heard loudly in the affected ear) occurs only in the presence of an open eustachian tube. On the other hand, in

4. Jago, J.: Brit. & For. Med.-Chir. Rev. **34**:175 and 496, 1867.

5. Toynbee, J.: The Diseases of the Ear: Their Nature, Diagnosis and Treatment, ed. 2, with a supplement by J. Hinton, London, H. K. Lewis, 1868, p. 447.

6. Politzer, A.: Wien. med. Wchnschr. **15**:1710, 1865.

7. Politzer,[3] p. 135.

8. Gruber, J.: Monatschr. f. Ohrenh. **2**:123, 1868.

9. Rudinger, N.: Monatschr. f. Ohrenh. **2**:84, 1868.

10. Lucae, A.: Arch. f. Ohrenh. **1**:96-106, 1864.

11. Weber, F. E.: Monatschr. f. Ohrenh. **2**:126, 1868.

12. Brunner, G.: Arch. Ophth. & Otol. **2**:107, 1870.

13. Brunner, G.: Ztschr. f. Ohrenh. **13**:286, 1883.

1873 T. F. Rumbold,[14] of St. Louis, wrote an excellent monograph on the eustachian tube in which he described in great detail the findings in 5 patients with continuously open eustachian tubes, including 1 with a perforated drum, and in his writings displayed a clear understanding of the phenomena associated with it. Similarly to Jago, he recorded many of the factors which tend to close the tube and thus make the symptoms disappear. His reports indicated that a continuously open eustachian tube is not as rare as Jago and Hinton had believed it to be. At about that time Hartmann,[15] in a series of papers culminating in a monograph published in 1879, reported on his studies, which form a foundation for knowledge of the physiologic nature of the eustachian tube in man. He reported that among the subjects he had studied a few had patent eustachian tubes and that the drum could be seen to move synchronously with respiration in such cases. Voltolini [16] in 1883 reported a similar observation. Sporadic reports of cases of continuously open eustachian tubes are found in the literature, such as by Buck,[17] McAuliffe,[18] Bezold [19] and Wagenhäuser.[20] Ostmann [21] studied the relation of atrophy of the fat pad adjacent to the cartilage of the eustachian tube to patency of the tube. While textbooks of otology written in the twentieth century rarely describe the clinical entity of the pathologically patent eustachian tube, Urbantschitsch [22] in his textbook published in 1890 clearly described the condition. A few paragraphs on the patent eustachian tube are to be found in the "Handbuch der Hals- Nasen- Ohrenheilkunde," [23] in which the rarity of the condition is pointed out. Perusal of the literature reveals little reference to this subject since the beginning of the twentieth century and little advance in knowledge of the normal physiologic state of the eustachian tube in man. It almost appears as if what was learned during the nineteenth century were minimized or overlooked by later generations of otologists. My observations, on the other hand, indicate

14. Rumbold, T. E.: The Function of the Eustachian Tube, St. Louis, Southwestern Book and Publishing Co., 1873.

15. Hartmann, A.: Experimentelle Studien über die Funktion der Eustachischen Röhre, Leipzig, Veit u. Comp., 1879.

16. Voltolini, R.: Monatschr. f. Ohrenh. **17**:1, 1883.

17. Buck, A. H.: Am. J. Otol. **2**:203, 1880.

18. McAuliffe, G.: New York Eye & Ear Infirm. Rep. **6**:116, 1898.

19. Bezold, F.: Arch. f. Ohrenh. **21**:232, 1884.

20. Wagenhäuser: Arch. f. Ohrenh. **21**:272, 1884.

21. Ostmann: Arch. f. Ohrenh. **34**:170, 1893.

22. Urbantschitsch, V.: Lehrbuch der Ohrenheilkunde, ed. 3, Vienna, Urban & Schwarzenberg, 1890, p. 196.

23. Denker, A., and Kahler, O.: Handbuch der Hals- Nasen- Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 6, p. 426, vol. 7, p. 99.

not only that, as Zöllner [24] and Shambaugh [25] recently pointed out, the continuously open eustachian tube is a distinct clinical entity but that it occurs more frequently than has been generally understood. The following studies on a number of patients are presented as additional evidence to clarify the clinical picture of the continuously open eustachian tube and to correlate it with the pathologic patency. Furthermore, in the latter half of this paper, observations pertaining to the normal function of the eustachian tube are described, along with the findings of recent investigators, and the pathologic function thus integrated with present concepts of the normal function.

REPORT OF CASES

CASE 1.—R. B., aged 28, came into the otorhinolaryngologic clinic on September 17, complaining of intermittent roaring in both ears, which made him hard of hearing. The sound was aggravated by forced respiration. Examination revealed nothing of note except that which pertains to the ears. Both drums were thinned and retracted, and the right was seen to move in and out synchronously with respiration. Insufflation of boric and salicylic acid powder into the right eustachian tube through a eustachian catheter caused the roaring sound to disappear, and on examination a week later no movement of the drum synchronous with respiration could be detected. A month later the patient was seen again and stated that the roaring had returned. Again the right drum was seen to move with respiration. Examination of the hearing showed that there was slight deafness in the left ear and a greater degree of deafness in the right. Two weeks later, when the roaring had disappeared, the hearing curves for the two ears were practically identical.

Three months later the patient was seen again and said that for a week during that period he had had symptoms in the opposite ear while the right ear was well. Then the symptoms disappeared from the left ear and returned to stay in the right. It was his impression that he had more symptoms when the weather changed suddenly from clear to cloudy. On examination the right drum was seen to move with respiration while the left was immobile. When the patient assumed the horizontal position, examination revealed that the right drum no longer moved with respiration. Examining the drum immediately after he assumed the erect posture again, one saw no movement. The pressure needed to force air through the tube into the middle ear was now tested (by a procedure to be discussed), and a pressure of 4 mm. of mercury exerted by the Valsalva method resulted in an audible thud marking the moment of entrance of air into the middle ear. Observation of the right drum then showed movements synchronous with respiration, which persisted. Observations on the left eustachian tube made with the patient in the erect position showed that a pressure of 30 mm. of mercury was needed to open it and allow air to enter the middle ear.

Comment.—The patient was in good health, and nothing found on examination of the ears, nose and throat explained the pathologic patency of the eustachian tube. Hearing tests revealed mild perceptive

24. Zöllner, F.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:137, 1936.

25. Shambaugh, G. E., Jr.: Continuously Open Eustachian Tube, Arch. Otolaryng. **27**:420 (April) 1938.

deafness. The hearing was worse in the right ear because sounds arising from the flow of air from the respiratory tract through the middle ear masked the hearing for sounds produced externally. While many factors have been found to effect the closure of a patent tube (table 1), the treatment that Bezold [26] described (the insufflation of boric and salicylic acid powder, four parts to one, into the tube by means of a eustachian catheter) is one of the most useful to the clinician.

Lucae [27] was the first to observe that changes in the position of the head, such as flexing the head on the chest, increase the resistance of the tube. Hartmann recorded the pressures needed to open the eustachian tube while the subject's head is in the erect, in the partly flexed and in the fully flexed position. He noted that the increased resistance did not leave when the patient again assumes the erect posture. My observations, on the other hand, of patients in the erect and in the reclining posture seem to indicate that when the erect posture is again assumed the resistance of the tube in almost all instances immediately returns to the previous figure for that posture.

The effect of changes in weather on the patient's symptoms may have been related to changes in barometric pressure, which probably were great enough to open the tube.

CASE 2.—A. E., aged 28, came to the neurologic department on Aug. 7, 1938, complaining of paroxysmal pain of three years' duration in the right cheek and jaw. A diagnosis of trigeminal neuralgia was made, and a retrogasserian neurectomy of the second and third divisions of the fifth nerve was carried out the next day. Postoperatively the patient was found to have involvement of the motor as well as the sensory division of the fifth nerve. When seen on August 26 in the otorhinolaryngologic department she gave a history of freedom from aural symptoms immediately after operation except for some earache and momentary clicking sounds in the right ear. On the day of her examination in the otorhinolaryngologic department she first began to notice that her own voice resounded loudly in her right ear and that she heard a rushing sound in that ear with each expiration. She did not hear any sound on inspiration. Examination revealed sensory changes in part of the external auditory canal and drum but nothing of note in the appearance of the drums except dulness. With a Siegle speculum, movements of the drum synchronous with respiration could be seen. The sound which the patient heard on expiration resembled that which was produced when the examiner blew across the mouth of a eustachian catheter properly placed in the right side of her nose (engaged in the mouth of the right eustachian tube) as it protruded from the nasal vestibule. The sound did not occur with the catheter in the left side. The results of the hearing test were not significant. At an examination four months later, the patient stated that she was still troubled with her right ear about once or twice a week for ten minutes at a time. She said that when she talked at that time her voice came out of the right ear. She found that she could relieve the sensation by making an inspiratory effort with her mouth and nose

26. Bezold, F., in Bezold, F., and Siebenmann, F.: Textbook of Otology, translated by J. Holinger, Chicago, E. H. Colegrove Co., 1908, p. 151.

27. Lucae, A.: Arch. f. Ohrenh. **3**:179, 1866.

closed. Her jaw deviated to the affected side when she opened her mouth widely, which indicated a persistence of the involvement of the motor division of the fifth nerve. When seen a month later she was still having symptoms but only on phonation of nasally produced sounds. These, she said, seemed to come out of her right ear. The drum, however, was not seen to move, even with deep respiration. When she assumed the horizontal position her symptoms disappeared.

Comment.—Politzer [28] reported in 1861 that the orifice of the eustachian tube in a dog increases after stimulation of the intracranial portion of the fifth nerve and that this effect is produced by the action of the tensor veli palatini muscle, which was probably paralyzed in this patient. The contention of many of the early otologists who studied the anatomy of the muscles of the eustachian tube that this muscle acts principally on the tube and not on the palate and should be called a dilator of the tube instead of a tensor of the palate is corroborated by the fact that in this patient, as in several similar patients, no definite change in palatal movement could be detected, while evidence of dysfunction (abnormal patency) of the eustachian tube was found. Several patients who did not show evidence of motor paralysis after section of the fifth nerve did not have symptoms of abnormal patency of the eustachian tube. The development of abnormal patency after operation on the gasserian ganglion in which the motor root was injured points to the tonic effect of the extrinsic muscles of the eustachian tube supplied by the fifth nerve (tensor veli palatini) as a factor in keeping the tube normally closed and offering definite resistance to its opening. It also emphasizes the need of reexamining the concepts pertaining to the function of all the muscles acting on the eustachian tube.

The fact that at the last examination symptoms were not present during respiration and respiratory movements of the drum were not observed while symptoms occurred on phonation of nasally produced sounds can be explained on the basis of the difference in the pressures created during respiration and during phonation. The pressures developed in the nasopharynx during respiration (about 5 mm. of mercury) were not sufficient to open the tube, while those developed by the production of some nasal sounds, such as "N" and "M," were sufficient to force the walls of the tube apart, thus sending the acoustic oscillations directly into the right middle ear, while the normal tube on the opposite side resisted these pressures. Furthermore, Hartmann showed that phonation was associated with a decrease in the pressure needed to inflate the tubes. Thus he found that during the pronunciation of "E," "I," "O" and "U" the resistance of the tubes dropped from 32 mg. of mercury to 12, as tested by Valsalva's maneuver.

CASE 3.—J. K., aged 30, came to the University of Chicago Clinics on Aug. 28, 1938, complaining of paroxysmal pain in the right side of the face for six years.

28. Politzer, A.: Würzburg. naturwissensch. Ztschr. **2**:92, 1861.

The neurologist diagnosed the condition as neuralgia of the second and third divisions of the trigeminal nerve and performed a retrogasserian neurectomy on August 30. The patient had been free of auditory symptoms before operation, and examination in the otorhinolaryngologic department revealed normal cochlear and vestibular function. The day after operation the patient noted high-pitched tinnitus, similar to a tone of 2,048 cycles, which lasted throughout the day, gradually decreased and was last heard for fifteen minutes fourteen days after operation.

A week after operation the patient noted that his voice resounded in the right ear and that he heard his breath sounds loudly in that ear. This lasted three hours, while the patient was out of bed. A minute after he lay on his back the symptoms were gone, and he could then resume the erect position without a recurrence of them. Another attack was noted two days later and the last attack thirteen days after operation. Examination a few days after operation and again eighteen days after operation revealed weakness of the right masseter muscle and deviation of the jaw to the right, indicating that the motor root also had been involved in the operative procedure. The patient was examined again three months later, when he stated that there was a resounding of his own voice in the right ear for short periods almost daily which was always relieved by lying down. He no longer heard his breath sounds loudly in that ear. The drum showed no movement during respiration. The pressure needed to inflate the tubes by Valsalva's method was now determined with the patient in the erect posture. It was found to vary between 2 and 10 mm. of mercury for the right ear during several observations and to be 30 mm. of mercury for the left, or normal, tube.

Comment.—This is another example of the effect of injury to the motor division of the fifth nerve on the extrinsic muscles of the eustachian tube which suggests that the normal tonus of the muscles attached to the tube and supplied by the fifth nerve is a factor in keeping the tube closed. The eustachian tube on several occasions shortly after operation remained open constantly and thus transmitted the sounds of respiration, as well as of the voice. Later there was some recovery of the tonus of the muscle of the eustachian tube, so that a definite positive pressure (though less than normally) was needed to force the tube open. The tonus was not sufficiently recovered to withstand the pressure of the nasal components of some of the voice sounds, and hence the patient experienced autophony in that ear.

CASE 4.—H. J., aged 52 and weighing 214 pounds (97 Kg.), came to the clinics complaining of deafness and tinnitus which had followed bilateral otitis media six years previously. Examination revealed scarred, retracted drums. In the left drum was a thin atrophic scar which could be seen to move freely with respiration, sinking in on inspiration and bulging on expiration. Hearing tests indicated advanced deafness of both the perceptive and the conductive type. Close questioning revealed that the noises in the left ear included "echoing" of the voice in that ear. Inflation caused the motion of the flaccid atrophic scar and the "echoing sound" of the voice to disappear temporarily. The patient was observed for several months, and it was noted that the mobility of the scar was often not seen at the beginning of the examination and that it appeared and disappeared at intervals of several minutes while he sat in the examining chair. Further, it was observed that the respiratory motion of the drum could be induced in about a minute by spraying the nose with a 10 per cent solution of cocaine hydrochloride

or a 0.25 per cent solution of neo-synephrin hydrochloride. Examination of auditory function for sound conducted through the external auditory canal and for sound conducted through the nose and the patent eustachian tube indicated that the patient heard better through the patent tube than through the external auditory canal.

Comment.—The patient's obesity was contrary to the general conception that pathologic patency of the tube develops only in patients who are emaciated. Superimposed on the combined deafness was the masking effect of respiratory sounds on the hearing in the right ear. The effect of vasoconstricting agents applied to the nose in reopening the eustachian tube may be explained by the direct effect of the vasoconstricting agents on the tissue at the mouth of the tube—in the nasopharynx.

The finding of a difference in hearing through the patent tube and through the canal, while not adequately controlled, is in line with observations on experimental animals which show that sound introduced directly into the middle ear causes a greater acoustic stimulus than the same sound introduced into the external auditory canal.

CASE 5.—Mrs. B. L., aged 28 and appearing well nourished, came to the otologic department on September 15 complaining that for many years she had noted intermittently the sound of rushing air in the left ear synchronous with respiration. She did not complain of impairment of hearing or tinnitus. More recently she had noted a similar sensation in the right ear. While there had been long intervals of freedom from these symptoms, since the beginning of summer the sound had been heard almost constantly in the left ear and had distressed her a great deal. Examination revealed normal drums. With the ausculation tube one could detect greater loudness, with reverberation, in the left ear as compared with the right when the subject was pronouncing "M" or "N." No respiratory motion of the drum could be seen, even with deep respiration. The roaring sounds in the left ear affected its hearing. This could be verified by threshold determinations with an audiometer when the sounds were gone (eustachian tube closed to air from the respiratory tract) and again when they were present. Figure 1 is a record of the threshold of hearing in the left ear when the tube was not open (interrupted line) and when the tube was open (solid line), during which time the rushing sounds of respired air were masking the hearing and raising the threshold. The entire frequency range is seen to be affected, but particularly the region of 512 cycles. The tubes were inflated with a mixture of four parts of boric and one part of salicylic acid powder. The patient returned in a week and reported that she was free of symptoms for the first time since the beginning of summer. She stated that for five days after the treatment the autophony recurred in the left ear for intermittent periods. It appeared about one-half hour after she arose and persisted until noon. Throwing the head forward while at the same time pressing the left side of the neck upward with the hand or lying down would cause the symptoms to disappear. If the erect posture were resumed, the symptoms recurred in fifteen minutes. For the last two days she had been free of symptoms. There were no pertinent laboratory, roentgen or medical findings except questionable residual apical tuberculosis diagnosed in 1934.

On examination three months later the patient stated that she had been free of symptoms until three weeks previously, when they had returned intermittently.

She could stop them as before by lying down or bending the head forward. She noted that a sleepless night or a strenuous day was likely to be followed the next day by a return of the symptoms.

A second insufflation of boric and salicylic acid powder into the left eustachian tube was carried out. The patient was seen again six weeks after this treatment. She stated that the treatment had had no appreciable effect on her symptoms after the first twenty-four hours and that, moreover, it was followed by tinnitus which had persisted, resembling a tone of 512 cycles. She stated that the symptoms arising from opening of the eustachian tube were more pronounced in the morning than in the afternoon and usually most evident during the first hour of the morning. The tube could be opened by blowing the nose or talking and would then remain open until the patient closed it. This she could do regularly as has been described. The pressures needed to open her eustachian tubes were now determined. It was found that in the erect position a pressure of 8 mm. of mercury opened the left tube, while a pressure of 20 mm. (the maximum that the patient could produce)

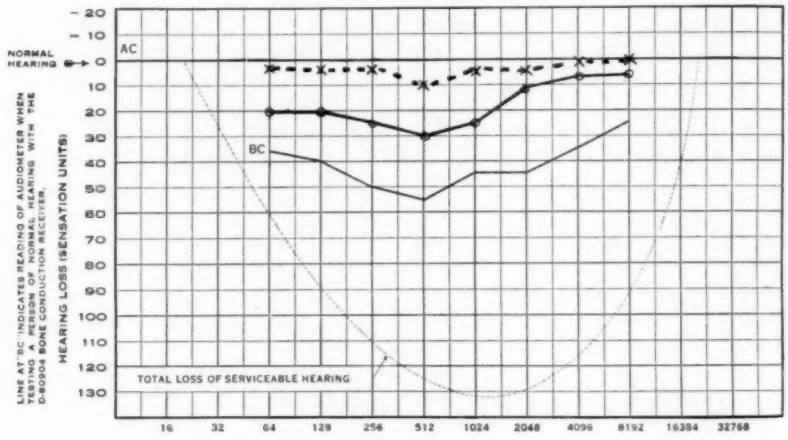


Fig. 1 (case 5).—Threshold curves of the left ear when the patient was breathing quietly. The solid line represents the curve made with the left eustachian tube open; the interrupted line, with the left eustachian tube closed.

failed to open the right. With the patient in the reclining position, neither tube could be opened at that pressure.

Comment.—The disappearance of the symptoms with closure of the tube on lying down or bending the head forward may be explained by slight venous or lymphatic stasis of the vessels in the mucous membrane lining the tube or by changes in tonus of the tubal muscles under those circumstances, which not only closed the tube but increased enormously the pressure needed to open it, or to force its walls apart. The patient illustrates the point that a patent eustachian tube may occur in the presence of a normal ear drum, which then cannot be seen to move synchronously with respiration. Warm summer weather has often been found by other observers, as by this patient, to be conducive to reopen-

ing of the tube and recurrence of symptoms. The recurrence of symptoms with fatigue is in line with the observations of Gyergyay,[29] who found that fatigue of the muscles about the tubes after a large number of acts of swallowing caused the tube to remain open. The observation that this patient's symptoms were more marked during the first hour of the morning also is in line with Gyergyay's findings that the eustachian tube opens most regularly during swallowing in the early part of the morning, after awakening.

CASE 6.—Mr. D., aged 40, came into the hospital with a serious valvular lesion of the heart of two years' duration for which he had been under close observation for many months. During that time he was referred to the otorhinolaryngologic clinic because of "head noises." His symptoms were typical of the patent eustachian tube and had been intermittently present for about three years. They were particularly noticeable in hot or cold weather. Coming into a warm room after being out on a cold day caused them to disappear. They always disappeared as soon as he lay down. They were not present on arising in the morning and did not appear until he had had his breakfast and had a bowel movement. If he lay down during the day they disappeared but reappeared rapidly on his arising. The right ear was more often involved than the left, and he was having more trouble during the past few months, because, as he thought, his cardiac condition was worse. Insufflating a mixture of boric and salicylic acid powder in both eustachian tubes, on September 13, was followed by marked improvement, so that two weeks later the left ear was well and the right ear was troubling him only occasionally. Pressure over the vessels of the neck had no effect. A diary of the periods through the day when symptoms appeared in the right ear, kept at this time by the patient, showed the following data:

Occurrence of Symptoms

9/13, 14, 15	No symptoms
9/16	10:16 to 10:43 a. m.
	10:45 to 11:10 a. m.
	11:20 a. m. to 12:25 p. m., when he went to bed
	4:10 to 4:30 p. m.
	6:20 to 7:10 p. m.
	8:02 to 8:14 p. m.
	8:26 to 8:55 p. m., when he retired
9/17	9:10 to 9:16 a. m.
	9:26 a. m. to 1:00 p. m., when he went to bed
	2:10 to 2:16 p. m.
	6:45 to 8:55 p. m., when he went to bed
9/18	Momentary return of symptoms several times during the day
9/20	12:15 to 1:00 p. m., when he went to bed
	2:00 to 2:41 p. m.
	3:08 to 3:14 p. m.
	3:25 to 4:10 p. m.
	4:50 to 5:05 p. m.

The mixture of boric and salicylic acid powder was insufflated into the right eustachian tube again on September 21. The patient was examined a number of

29. Gyergyay, A.: Monatschr. f. Ohrenh. **66**:769, 1932.

times when the symptoms were present. Both drums were thin and flaccid. They could always be seen to move synchronously with respiration when the symptoms were present. With an auscultation tube in the external auditory canal on the affected side, one could hear "M" and "N" reverberate loudly and a distinct blowing sound synchronous with respiration. When seen on November 16 the patient stated that he had been free of symptoms, and examination corroborated this. At 9:30 a. m. the right side of the nose was sprayed with a 0.25 per cent solution of neo-synephrin hydrochloride. At 9:32 the symptoms returned in the right ear and remained for six minutes, after which they disappeared, on use of the voice. On November 22 the symptoms were again made to reappear by spraying the nose with a 0.25 per cent solution of neo-synephrin hydrochloride. They disappeared as soon as the patient began to speak. A 10 per cent solution of cocaine hydrochloride sprayed into the nose had an effect similar to that of neo-synephrin hydrochloride.

On September 22, while the symptoms were present, it was possible to obtain objective evidence that the acoustic energy entering the affected middle ear was greater than that on the unaffected side when the patient pronounced the letter "M," by recording the sounds on a General Electric oscillograph with the aid of two microphones and suitable amplification. One microphone was connected by rubber tubing to an olive tip inserted into the external auditory canal. The other

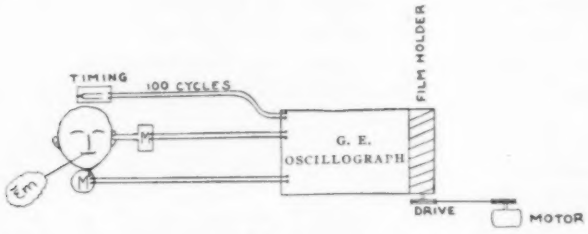


Fig. 2.—Diagram illustrating the method of recording "autophony." *M* represents a microphone connected by a suitable tube and tip with the external auditory canal; M_1 a microphone at the patient's mouth to record enunciation of the letter "M." The currents from the microphones and the oscillator are conducted through amplifiers, not illustrated, and recorded on the General Electric oscillograph.

was at the patient's mouth. When he pronounced the letter "M," the sound pattern at the mouth was recorded in one tract, and at the same time the sound pattern transmitted from the nasopharynx through the eustachian tube and the drum and out along the external auditory canal was recorded on another tract. A timing wave of 100 cycles per second was recorded at the same time. The general arrangement for this experiment is illustrated in figure 2.[30]

It will be noted on examining the record (fig. 3*A*) that the first part (ĕ) of the vocalization of "M" is not transmitted through the eustachian tube. This is to be expected, since it is not a nasally produced sound but is made by forcing air through the open mouth with the tongue held near the hard palate. As the "m-m" is formed, by closing the mouth and forcing air through the nose, the acoustic energy is transmitted not only through the nose but out along the open eustachian tube from the nasopharynx. This transfer of energy to the middle ear through the tube is a gradual one, as seen by the shape of the sound tract.

30. This experiment and the one illustrated in figure 6 were done with the aid of Dr. T. Case, of the department of neuropsychiatry.

The sound tract of the affected ear is in contrast with that of the unaffected ear (fig. 3*B*). Here one sees no such transfer of acoustic energy. Only a small amount is picked up, at a fairly constant level, and this comes from the general vibration of the skull and the transmission of energy to the microphone in the ear through the air and tubing from the sound produced at the subject's mouth.

Objective evidence that symptoms always disappeared when the patient was in the prone position was obtained on several occasions. The pressure needed to open the eustachian tube when the patient was symptom free and in the erect position was compared with that needed when the patient was lying down. It was found that while a pressure of 2 to 8 mm. of mercury could open one tube

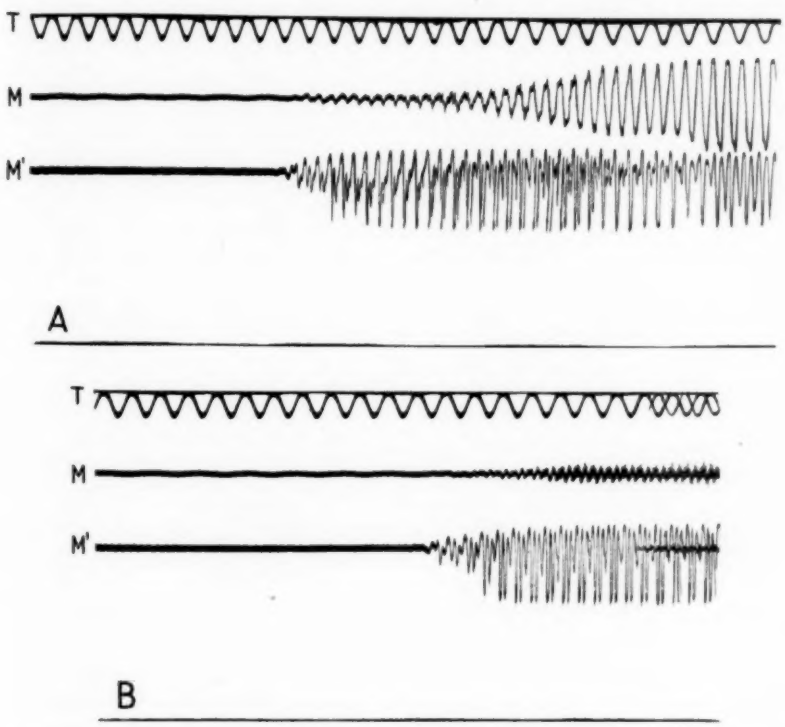


Fig. 3 (case 6).—*A*, record of "autophony" when the patient pronounced the letter "M" while the eustachian tube was open. *B*, record made under conditions identical with those of *A* but from the unaffected ear. *T* represents a timing wave of 100 cycles per second; *M*, the sound tract from the microphone connected to the external canal; M^1, the sound tract from the microphone at the mouth of the patient.

when the patient was in the erect position, the same tube could not be opened with a pressure of 40 mm. (the maximum that he could produce) when he was in the prone position.

CASE 7.—Mrs. V. E., aged 21, came into the University of Chicago Clinics complaining that for a number of years she had heard noises in both ears and also experienced fulness in the ears. The right ear had begun to pain about five

months previously. Examination gave essentially negative results except for the ears. Both drums were thinned and retracted, and the right could be seen to move back and forth synchronously with respiration. Hearing was normal. With the auscultation tube one could hear respiratory sounds as well as the resounding of nasally produced sounds, such as "M" and "N." The right eustachian tube was insufflated once a week for four weeks with the mixture of boric and salicylic acid powder, with only temporary relief of symptoms. Examination two weeks after the last treatment revealed both drums to be moving with respiration. The patient complained of fulness in the ears, which was relieved by holding the breath. Moving her head into different positions had no effect on the respiratory mobility of the drum. She stated that speaking in an ordinary low conversational voice caused no discomfort. Introduction of a drop of a 20 per cent solution of silver nitrate into the right eustachian tube seemed to have no beneficial effect, and examination a week later again showed respiratory mobility of both drums. As in the other patients, the sound produced by inflating the tubes with a catheter and Politzer bag was distinctly abnormal, there being no resistance to the air stream. However, it resulted in a cessation of the respiratory movements of the drum for several minutes at a time. The patient's hearing was tested through the nasal fossae with both canals filled with wet cotton and after the right tube was closed temporarily by inflation. The sound was conducted through a glass nasal suction tip connected to a funnel placed over the receiver of a 2-A audiometer. The difference in hearing through the nasal fossa on the side with the tube open and through that on the side with the tube closed is illustrated in figure 4 *A*. When the hearing was tested again, after both tubes were closed, this difference had disappeared (fig. 4 *B*).

The patient was seen again two months after her last treatment and said she had obtained no relief and also that she now noted a pulsating tinnitus unrelated to her breathing or phonation and aggravated by exercise. Both drums were found to be moving synchronously with respiration. This movement could still be observed when the patient was in the reclining position, a marked contrast to the findings in the other patients studied, indicating greater patency of the tubes and showed by only one other patient, not described here. A Mrs. B., whose symptoms developed during pregnancy, stated that they were present even when she was in bed, and on examination movement of the drum synchronous with respiration was noted in the erect and in the prone position.

Case 8.—Dorothy L., aged 26, had been suffering from ulcerative colitis for ten years and was emaciated. For three years she noted "stoppage" of her ears from ten to thirty minutes at a time. An abdominal operation done about six weeks before examination in the otorhinolaryngologic clinic was followed by a return of the patient's aural symptoms for five days, but only while she was sitting up in bed. She stated that at this time she experienced a sensation of fulness in the ears and that when she spoke her voice sounded as if she were speaking in a barrel. Her own voice sounded loud to her, but her listeners stated that her voice was low. These symptoms disappeared when she lay down and always reappeared when she sat up. She could make them disappear for a few moments at a time by striking her ears with the hands. All symptoms disappeared in six days. After a second operation on December 2 the symptoms returned on the patient's sitting up and continued intermittently. The hearing was normal when she was examined on December 20, at which time she was free of symptoms. The drums appeared normal. On December 22 the patient noted a return of symptoms and was sent from her hospital bed to the clinic in the sitting position. With the auscultation tube, breath sounds could be heard distinctly in both ears. Nasally

produced sounds were heard loudly by the examiner through the auscultation tube. The posterior part of the drum was seen to move synchronously with respiration. Threshold determinations with the 2-A audiometer revealed a loss of hearing, most marked for 512 cycles (fig. 5), similar to the loss noted in Mrs. L. When the patient assumed the horizontal position the symptoms disappeared. Several determinations, when the patient was free of symptoms, of the pressures needed to open the eustachian tube indicated that while the right and left tubes could be

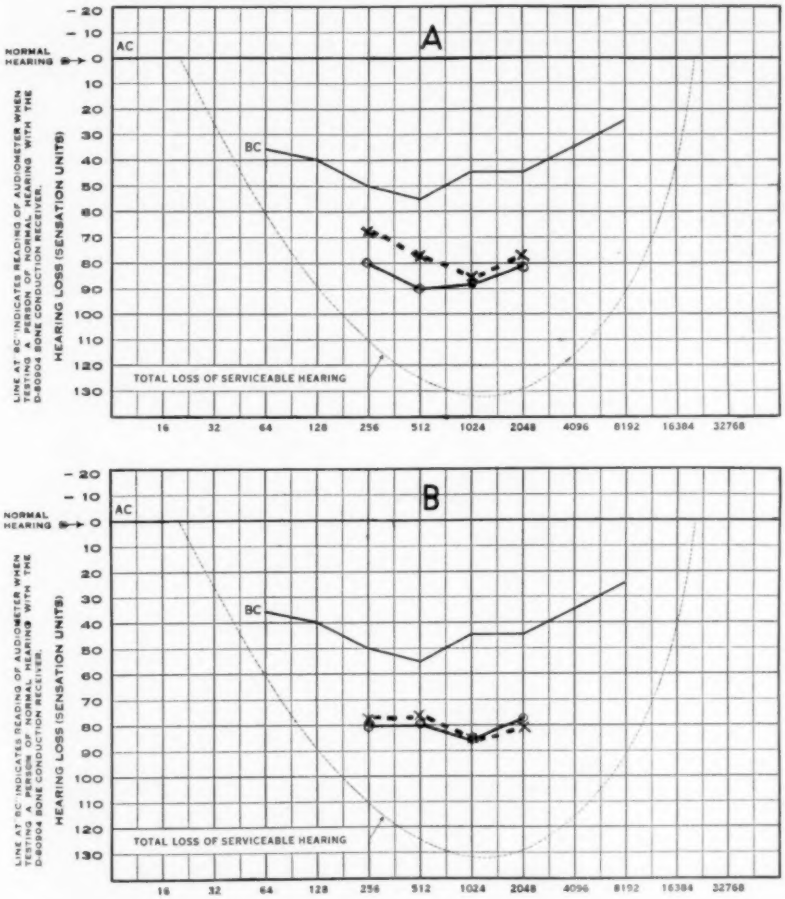


Fig. 4 (case 7).—Threshold curves for sound delivered to the nose (*A*) when the left eustachian tube was open and (*B*) when both eustachian tubes were closed. The sounds were introduced with the aid of a glass nasal suction bulb connected with the receiver of a 2-A audiometer by a funnel and rubber tubing into the right or the left nostril. The solid lines show the values with the bulb in the right nostril; the broken lines, with the bulb in the left nostril. The threshold was raised for the side on which the tube was open.

opened by a pressure of 2 and 18 mm. of mercury, respectively, when she was erect a pressure of 40 mm. (the maximum that she could produce) was insufficient to open either tube when she was prone.

When the patient was symptom free (on Jan. 9, 1939) an experiment was carried out to determine in what way and for how long the eustachian tube opens during the act of swallowing and transmits sound. The experimental setup is diagramed in figure 6. In preliminary studies it was found that a distinct train of muscle action potentials for the act of swallowing could be recorded by leads from two silver electrodes placed on the neck over each end of the hyoid bone. A 500 cycle tone of moderate intensity was carried from a suitable oscillator through tubing and a glass bulb to the subject's nose. A microphone attached by

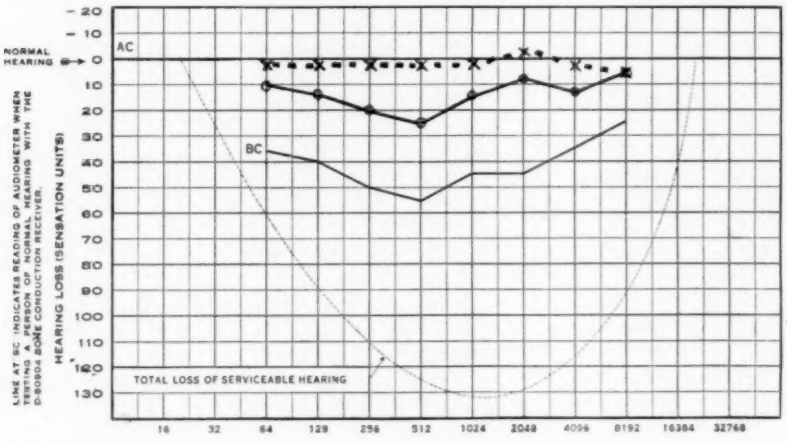


Fig. 5 (case 8).—Threshold curves of the right ear when the patient was breathing quietly. The solid line represents the curve made with the tube open; the interrupted line, with the tube closed.

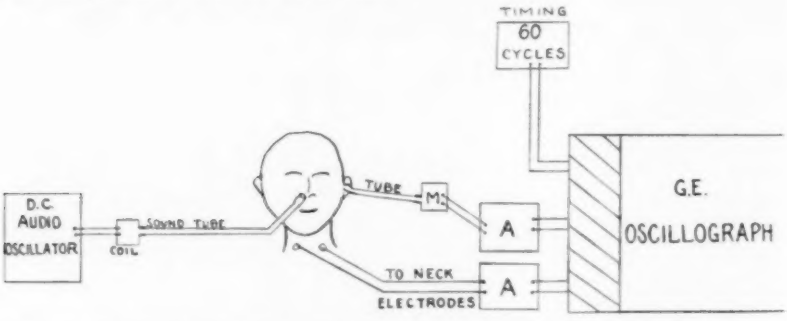


Fig. 6.—Diagram representing the experimental setup to determine transmission of sound through the eustachian tube during the act of swallowing. *M* indicates the microphone; *A*, the amplifier.

tubing and an ear tip and connected to an amplifier was used to detect the sound spectrum in the external auditory canal. A timing wave of 60 cycles per second was used as a reference. The activity from the three tracts (muscles of the neck, external auditory canal and timing mechanism) was recorded simultaneously on bromide paper with a General Electric oscillograph. In this way any marked change, though transient, in the acoustic energy transmitted to the middle ear from

the nose through an open eustachian tube could be recorded, analyzed and integrated with the act of swallowing.

Several subjects were studied in this way with varied results. The findings for the patient under discussion are presented as illustrative of a type of response obtained by the method described. The record illustrated in figure 7 is representative of a number obtained from this patient, showing the muscle action potentials in tracing *D* and the acoustic spectrum from the canal in tracing *M*, along with the tracing of the timing wave A of 60 cycles per second (*T*). One notes that the duration of the act of swallowing as measured by the muscle action currents is about one and three-fifths second. In the sound tract from the canal, one notes, about two fifths of a second after the onset of deglutition a large spike suddenly appears followed by an oval spread on the base line. If this area is examined with a hand lens one can detect the 500 cycle component. The initial sharp spike probably represents the click initiating the opening of the tube, as the walls of the tube separated. The entire cycle of opening of the eustachian tube is seen to last about three tenths of a second. The spikes appearing periodically in the

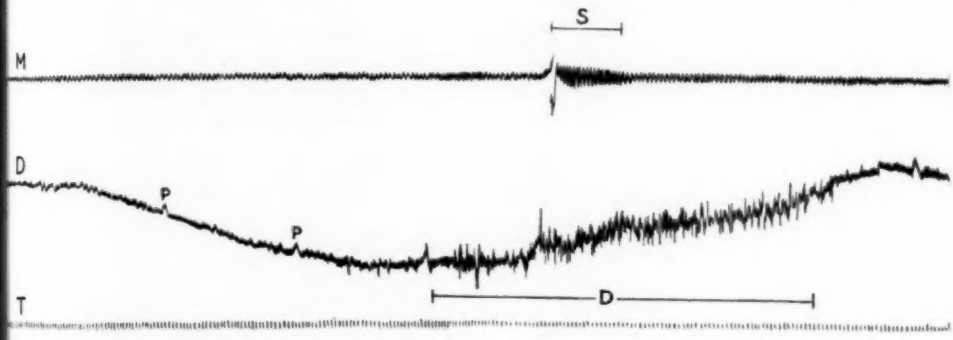


Fig. 7 (case 8).—Record showing the relations between swallowing and opening of the eustachian tube as affecting transmission to the middle ear of a tone of 500 cycles entering the nasal fossa. *T* represents a timing wave of 60 cycles per second; *M*, the activity of a microphone connected to the external auditory canal; *D*, the tract for muscle action potentials tapped by electrodes on the neck. The duration of the complex representing the act of swallowing is indicated by the bracketed line, *D*. The complex associated with the opening of the eustachian tube is indicated by the bracketed line *S*. *P* represents the disturbance at the electrodes on the neck synchronous with the pulse.

muscle action response are synchronous with the subject's pulse. The regular undulations in the tracing from the microphone are 60 per second.

Case 9.—Mr. D. M., aged 47, came into the clinics complaining of noises in the right ear. The sounds were noted while talking, and it felt as if he were "talking in a barrel." His voice reverberated in his right ear only. The symptom was intermittent, lasting as long as a full day. He could also hear the rushing of air in the affected ear with each respiratory effort. On examination when he was symptom free his hearing was found to be normal, as was the appearance of the drum. He was seen three months later, when he stated that he had been free of symptoms except for ten minutes immediately after his last visit.

Comment.—The patient's record is introduced as representative of those of a group of patients examined in the clinic, sometimes with

negative results but usually with evidence of pathologic patency of the eustachian tube as tested by the manometer. On carefully questioning these patients one can reveal that their complaints are definitely related to periods of varying duration when their eustachian tubes remain open.

CASE 10.—Retrogasserian neurectomy was performed on the right on August 1 on Mr. D., aged 34, for trigeminal neuralgia. On examination in the otorhinolaryngologic clinic before he left the hospital the patient stated that since the operation he noted that when he blew his nose his right ear stopped up. This also occurred when he drank something cold. Immediately thereafter his voice resounded in that ear for about fifteen minutes. When he was seen again six months after operation he stated that these aural symptoms had persisted for about four months and had then disappeared except for three or four periods of about fifteen minutes each during the last two weeks. He said that he had never had symptoms while lying down. The patency of his eustachian tubes was then determined with Valsalva's maneuver, and it was found that with the patient in the erect position a pressure of 12 mm. of mercury was sufficient to open the right tube (on the side of the operation), while a pressure of 24 mm. was needed to open the left tube. While he was in the reclining position neither tube could be opened by the maximum pressure that the patient could produce (50 mm. of mercury). Again, after he arose, the right tube was found to inflate under a pressure of 14 mm. of mercury. By directly observing his drums for bulging during the Valsalva maneuver one could see evidence of air entering the right middle ear when the pressure in the tube was only 4 mm. of mercury. On the normal side (the left) a pressure of 20 mm. was needed to produce a noticeable bulge of the tympanic membrane.

Comment.—The findings represent a mild form of the pathologically patent eustachian tube.

OBSERVATIONS

Table 1 lists the factors which have been found by observations on the patients who have been discussed and reported by previous investigators to effect closure of the eustachian tube with relief of symptoms and the factors which tend to cause the tube to reopen with recurrence of symptoms.

The ability to recognize and define pathologic states of the eustachian tube is hindered by inadequate knowledge of its normal physiologic state. The investigations in the nineteenth century by such keen observers in this field of otology as Toynbee, Jago, Politzer, Hartmann and Rumbold laid the groundwork for understanding of its function, and little has been reported since then that has enhanced the knowledge of its physiologic nature and pathologic conditions. However, in recent years some renewed interest in this field has been evidenced by the investigations of Gyergyay,[29] Zöllner [31] and Armstrong and Heim.[32]

31. Zöllner, F.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **42**:287, 1938; Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:137, 1936.

32. Armstrong, H. G., and Heim, J. W.: Effect of Flight on the Middle Ear, J. A. M. A. **109**:417 (Aug. 7) 1937.

Besides my study of the patients with pathologically patent eustachian tubes, a large number of observations pertaining to the patency of the eustachian tube were made on normal and diseased ears to corroborate the findings of Hartmann, to reexamine the concepts of the function of the tube and to formulate a background for my observations on the patients described.

The eustachian tube is normally closed, and its walls are held in apposition by the tonus of the tubal muscles. Hartmann found that subjecting a person in a pressure chamber to a pressure of as much as 130 mm. of mercury (when the pressure was on the outer surface of

TABLE 1.—*Factors Affecting the Patency of the Eustachian Tube*

Factors Causing Closure of the Patent Eustachian Tube	Factors Causing the Pathologically Patent Tube to Open and Remain Open
Inspiratory effort with mouth and nose closed (Jago)	Summer and autumn (Jago)
Hearty meal	Nervousness
Reclining posture	Increase in activity of the heart
Bending head forward on chest (Lucae)	Fatigue, as from sleepless night or strenuous activity
Catheterization and inflation of the tube	Erect posture
Application of silver nitrate to mouth of tube (Rumbold)	Loss of weight
Insufflation of boric and salicylic acid powder (4 parts to 1)	Hot weather
Coming into warm room on cold day	Being out in cold day
Strong pressure on tragus	Cold drink
Constant electric current with canal filled with saline solution	Spray of 0.25 per cent solution of neosynephrin hydrochloride in nose
In presence of perforated drum (Rumbold)	Spray of 10 per cent solution of cocaine hydrochloride in nose
(*a*) Drop of water in ear	Deep respiration
(*b*) Pledget of cotton soaked with glycerin water	Pronouncing nasally produced sounds, such as "M," "N," "ng"
Bougie in eustachian tube	Coughing, belching, sneezing, blowing the nose
Filling external canal with saline solution	Dysfunction of tubal muscles after injury to motor division of fifth nerve in retrogasserian neurectomy
Gargling	Stooping, yawning
Speaking	Loss of tissue of the tube, as in syphilis or carcinoma
Inflation of eustachian tube with zinc sulfate solution	Beginning or "dry stage" of acute infection of upper part of respiratory tract (Rumbold)
Injection of paraffin about the tube (Zöllner)	

the drum, pain in the ears was noted) does not cause the tube to open. Only by the subject's swallowing does the tube open and pain disappear when the pressures on both sides of the drum are equalized. On the other hand, in the negative pressure chamber, Hartmann found, movement of air from the middle ear through the tube occurs in small puffs which sometimes cause a "flutter" of the drum and begin with a negative pressure of about 30 mm. of mercury.

The studies by Armstrong and Heim, from the Wright Air Field, showed that a decrease of pressure of 3 to 5 mm. of mercury, corresponding to an ascent of 110 to 180 feet (33.5 to 55 meters), results in a sense of fulness in the ears and bulging of the ear drums. When a negative pressure of 15 mm. of mercury is obtained, corresponding to an elevation of 500 feet (152 meters), a sudden, annoying click is

heard and felt, with relief of the sense of fulness and pressure and a return of the drum to its normal contour, as the positive pressure in the middle ear forces the walls of the eustachian tube apart. As negative pressure is increased further (ascent continues), clicks occur about every 435 feet (132.5 meters). This represents a change in pressure of 11.4 mm. of mercury at sea level but of only 3.5 mm. at 40,000 feet (12 kilometers). That diminishing changes in pressure open the tube as ascent continues was explained by the authors by the increased permeability of the rarefied air. From their observations they concluded that a positive pressure of 15 mm. of mercury in the middle ear is needed to open the tube. Then it remains open until the pressure is reduced to 3.6 mm., when it closes again, leaving an excess of pressure of 3.6 mm. in the middle ear.

Under positive pressure, they found, the tube would not open when swallowing was avoided, even at a pressure of 470 mm. of mercury, at which the drum would rupture. They compared the action of the eustachian tube to that of a "flutter valve." They found further that

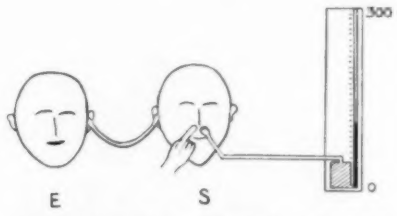


Fig. 8.—Diagram representing the method for the quantitative determination of the patency of the eustachian tube during Valsalva's maneuver. *E* represents the examiner, whose ear is connected to the ear of the subject (*S*) through the diagnostic tube. The subject's nose is connected by tubing and a glass tip to a common blood pressure manometer. With one nostril closed the subject exhales forcibly into the tube, thereby forcing the column of mercury upward. The examiner watches the column of mercury as he listens for the distinct "lub" which marks the moment that the tube opens and the drum is forced outward.

when the pressure in the middle ear is 90 mm. of mercury below the atmospheric pressure, as in rapid aeronautic descent, the muscles of the eustachian tube in swallowing can no longer overcome the pressure which keeps the walls of the tube in apposition and the resultant discomfort in the ears cannot be relieved.

Hartmann's experiments showed that much lower positive pressures (20 to 40 mm.) were needed to inflate the ear by the Valsalva maneuver. He determined this by direct observation of the drum for the onset of bulging, at which time the pressure in the connected manometer was noted. This was the pressure needed to open the eustachian tube and to overcome the resistance of the tubal muscles. The observation can easily be checked by the otologist in the following simple way:

The mercury manometer of a blood pressure apparatus is connected by rubber tubing to a glass bulb nasal suction tip. The subject puts this firmly into one nostril and blows into it while he closes his other nostril with a finger. The examiner listens with an auscultating tube as he watches the level of the mercury column. The general arrangement is diagramed in figure 8. Usually the sound produced in the normal ear by this procedure is a faint, low-pitched "lub," which, nevertheless, can be easily recognized by the examiner, due to the sudden outward displacement of the drum (Politzer). When the drum is perforated a squeaking sound is heard, which usually is loud enough

TABLE 2.—*Resistance of the Eustachian Tube During Valsalva's Maneuver in a Representative Group of Normal Subjects when Erect*

Name	Sex	Age	Pressures Needed to Inflate Tube, Mm. of Hg.	
			Right Ear	Left Ear
V.	M	32	18	..
L.	F	59	22	22
D.	F	10	20*	20*
P.	M	17	18	22
D.	M	38	32	..
G.	F	14	..	30*
K.	M	49	45*	45*
C.	F	10	..	20
B.	M	50	27	..
B.	M	23	18	..
K.	M	40	..	40
K.	M	50	30	30
B.	M	28	..	30
L.	F	28	..	20
K.	M	22	30	30
J.	M	7	20	..
S.	M	69	40*	25
P.	M	38	18	18
K.	M	44	26	22
R.	F	39	22	22
D.	M	25	..	22
F.	M	58	25	..
M.	M	23	29	32
K.	M	41	55	55

* Maximum pressure, developed without opening tube.

to be recognized without an auscultating tube. The examiner may also observe the drum for bulging as Hartmann described. This requires an assistant who will note the pressure in the manometer when the examiner signals the appearance of changes in the drum. One finds that a certain minimum pressure is needed and that above that great variation occurs. This minimal pressure is about 20 mm. of mercury with the subject in the erect posture. Some of the pressures needed to open the eustachian tube in normal ears are recorded in table 2. These pressures are in contrast with those found in the patients described, with abnormally patent tubes, which are listed in table 3. The resistance of the tube is decreased by exercise. As an example, a normal subject who was able to open his tube with a pressure of 20 mm. of mercury found that after jumping up and down ten times in three

minutes he could open his tubes with a pressure of only 10 mm. The pressure required returned to 20 mm. after five minutes. This finding of decreased resistance of the tube with exercise holds true for diseased

TABLE 3.—*Resistance of the Eustachain Tube During Valsalva's Maneuver in Some of the Patients Described, Measured when the Walls of the Tube Were in Apposition* *

Name	Sex	Age	Pressures Needed to Inflate Tube, Mm. of Hg.	
			Right Ear	Left Ear
B.	M	28	4	..
K.	M	30	2	..
L.	F	26	2	..
D.	M	40	2	10
L.	F	28	8	..
E.	F	28	2	2
D.	M	34	12	..

* The figures varied from time to time in each subject but were always below the average normal.

TABLE 4.—*Effect of Exercise* * *on the Resistance of the Eustachian Tube During Valsalva's Maneuver*

Name	Sex	Age	Pressures Needed to Inflate Tube, Mm. of Hg.	
			Before Exercise	After Exercise
D.	M	38	28†	20
A.	M	32	20	10
F. Right	M	30	30	22
Left			28	22
E. Right	F	38	35†	20
Left			30	10
W. Right and left	F	15	35	30
K. Right	M	49	35	30
B. Left	M	28	30	27
F. Right and left	M	30	50†	20
G. Right	F	37	40†	20
S. Left	M	60	25	20
Right			40†	30
P. Right and left	M	38	18	10
D. Left	M	40	22	18
P. Right	M	17	18	15
Left			22	18
F. Right	M	58	25	15
K. Right and left	M	41	55	48
F. Right	M	32	30	22
Left			28	22
L. Right	F	50	22	20
Left			22	16
S. Left	M	51	35	20
Right			30	22
O. Left	M	28	20	10

* Bending forward ten times.
† Maximum pressure, developed without opening tube.

ears as well as for normal ears, and some instances are recorded in table 4. Assuming the horizontal position decreases the patency of the tube or increases its resistance to pressure as tested by Valsalva's

maneuver and by Toynbee's maneuver, in other words, to pressure directed toward the middle ear and to pressure directed from the middle ear to the nasopharynx. The increase may be great—as in the patient with a pathologically patent tube who could open it with a pressure of 2 mm. of mercury in the erect position and could not open it with a pressure of 40 mm. in the reclining position. The increased resistance of the tube when the subject is in the reclining position is found in

TABLE 5.—*Effect of Assuming the Prone Position on the Resistance of the Eustachian Tube During Valsalva's Maneuver*

Name	Sex	Age	Pressures Needed to Inflate Tube, Mm. of Hg.	
			Subject Erect	Subject Prone*
F. Right	M	32	30	45
Left			28	45
L. Right and left	F	50	22	55
M. Right	M	23	29	45
Left			32	45
F. Right	M	58	25	60
P. Right	M	17	18	50
Left			22	50
D. Left	M	40	22	30
P. Right and left	M	38	18	40
S. Right and left	M	69	25	40
G. Right	F	30	20	50
F. Right	M	30	20	50
Left			30	50
L. Right	F	23	10	30
Left			2	30
D. Left	M	40	8	40
Right			10	40
K. Right and left	M	30	2	40
B. Right	M	28	4	40
L. Left	F	28	8	20
W. Right and left	F	15	35	50
K. Left	M	30	35	45
G.	F	..	20	50
H	M	25	20	70

* Maximum pressures, developed without opening tube. No subject was able to inflate his eustachian tube while prone.

diseased ears as well as in normal ears. Some of the changes in pressure are recorded in table 5. Contrary to the generally accepted opinion, Gyergyay found that the tube does not open regularly and to the same degree with each act of swallowing or yawning. He found that the tube opens only occasionally during swallowing. There is a great deal of variation from subject to subject and in the same subject. He found that on the subject's awakening in the morning the tube opens most regularly during swallowing, as tested by Politzer's method of determining the change in loudness of a sound from a source at the nose during the act of swallowing. He stated the belief that when differences in

pressure between the air in the middle ear and the external air are sufficiently great the tube opens during swallowing. This seems reasonable when one finds that with the body in the horizontal position, as during sleep, the eustachian tube is practically impermeable. Thus, on the subject's arising the pressure of the air inclosed in the middle ear may correspond to the barometric pressure of the night before, while the barometric pressure may be changed considerably by the following morning. In the Chicago area the average fluctuation in barometric pressure is about 3 mm. of mercury, with greater and more rapid changes occurring in the winter than in the summer. These changes in barometric pressure when the eustachian tube is closed for a long period, as during sleep, may create sufficient inequalities in the pressure within and without the middle ear to impair function noticeably in a person who is already hard of hearing. Usually, however, the differences in pressure between the air in the middle ear and the outer atmosphere are sufficiently great to overcome the small resistance (0.5 to 4 mm. of mercury, Zöllner) of the tube during swallowing when the head is erect, and therefore equalization between the pressures within and without the middle ear takes place through the tube after getting out of bed.

A number of normal persons were examined in a manner similar to that of Politzer, by the use of the receiver of an audiometer connected by a funnel and glass bulb to the subject's nose. The observations of the degree and duration of the changes in loudness are in accordance with Gyergyay's observations. Gyergyay found also that as the muscles of the eustachian tube become fatigued by frequently repeated acts of swallowing (140) the tube becomes more and more patent as tested by the transmission of sound from the nose, until, at the end, the tube is open so that sound is constantly transmitted through it. As has been mentioned, Zöllner found that in 200 normal young adults tested for the aviation service a certain positive pressure was needed to open the tube even during swallowing. This was determined by observing the drum for motion with an aural microscope. In 83 per cent of his normal subjects a pressure of 0.5 to 4 mm. of mercury was needed to open the tube. (In the rest a still higher pressure was required.) Comparing these pressures with those found necessary to open the tube when the palate is at rest, one sees that at least a great decrease in the resistance of the tube takes place during the act of swallowing. In this way nature has provided an excellent mechanism to provide proper aeration of the spaces in the middle ear without subjecting the delicate auditory mechanism to the pressures prevailing in the nasopharynx during respiration, phonation or even sneezing or blowing of the nose. Only when the respiratory stream through the nasopharynx is suspended and the nasopharynx is sealed from the pharynx by the contracted palate does the resistance of the tube become low enough to permit

equalizations of the small differences in pressure between the middle ear and the external environment which normally develop during long periods of closure of the tube with changes in barometric pressure at the same level and with changes of elevation. The pressures tending to open the tube which are normally put on the walls are not sufficient to accomplish an opening. Such normal pressures are those which arise in the nasopharynx on phonation of nasal sounds, such as "ing," "M" and "N," on quiet respiration through the nose or, more markedly, on deep respiration through the nose, sneezing or coughing. When any of these smaller pressures consistently opens the tube and acoustic pressure or static pressure is thereby transmitted directly to the middle ear the tube is not functioning normally (is abnormally patent). The act of forcibly blowing the nose creates an abnormally high pressure (up to about 100 mm. of mercury) in the nasopharynx and is therefore likely to force the walls of the eustachian tube apart even in the normal person, but usually the nose is blown in such a way as not to create such a high pressure, and therefore the tube does not open. In the patient with an open eustachian tube blowing the nose, sneezing or belching may cause severe pain because of pressure on the drum. When all the changes in acoustic and static pressure arising in the nasopharynx are instantly and continuously transmitted to the middle ear through the eustachian tube, the tube is continuously open. Many persons, of whom I am one, can voluntarily open the eustachian tube and experience the symptoms of the patient with a continuously open tube. It is accomplished by contracting the palatal muscles in a way similar to that which occurs when stifling a yawn and is always initiated by a loud "click" heard in the ear. This "click" probably arises from the sudden separation of the walls of the tube. As soon as the palate drops the tube closes. While it is open the currents of respiratory air in the nasopharynx extend into the middle ear and can there be heard by the subject as a loud, low-pitched, blowing sound, synchronous with respiration. It can be heard also by an observer with the aid of a diagnostic tube. With nasally produced sounds acoustic pressure is transmitted directly to the middle ear, and thus the subject's ear is more intensely stimulated than by the same sound reaching the ear through the auditory meatus. Experimentally it has been shown [33] that sound introduced directly into the middle ear causes greater acoustic stimulation as measured by the acoustic tensor reflex than a similar sound introduced into the external auditory canal. This may be a factor in explaining the loudness of the sounds heard in the affected ear by a

33. Kobrak, H.; Lindsay, J. R., and Perlman, H. B.: Acoustic Stimulation of the Inner Ear by the Application of Sound into the Cavity of the Middle Ear, Arch. Otolaryng. **23**:39 (Jan.) 1936.

person with a patent eustachian tube. The person's respiratory sounds under these circumstances have a masking effect on the ear which interferes with the hearing—especially of sounds at threshold—but the hearing otherwise may be unaffected.

When the drum is normal no movements of it that are synchronous with respiration can be seen. When the tube remains patent for many years, changes in the drum and the mucous membrane of the middle ear due to their exposure to these abnormal pressures may eventually occur. If the drum is atrophic or flaccid or has a thinly healed perforation, the atrophic scar or the entire atrophic or flaccid drum can be seen to move synchronously with respiration. On inspiration there is an indrawing and on expiration an outward displacement of the drum. Because of the difficulty of many persons in clearly expressing the sensory phenomena that they experience, it is necessary for the examiner to question the patient carefully to elicit the symptoms which are found when the eustachian tube is abnormally patent, or remains open continuously.

SUMMARY

The basic facts pertaining to the normal physiologic state of the eustachian tube which have been brought out sporadically during the last hundred years should be correlated in an attempt to develop a rational basis for functional tests, particularly such as can be carried out easily without the use of special or elaborate equipment.

Emphasis is placed on the simple, practical method for quantitative determination of the patency of the eustachian tube by the use of the manometer of the common blood pressure apparatus, and a diagram is presented to illustrate this method, which, together with observation of the drum during Valsalva's and Toynbee's maneuvers, permits easy determination of the function of the eustachian tube.

As tested by this method during autoinflation, or Valsalva's maneuver, the tube offers a minimal resistance of about 20 mm. of mercury in a subject in the erect posture, yet a much greater resistance (60 +) appears to be compatible with normal auditory function.

The resistance of the tube is regularly decreased by exercise.

The resistance of the tube is greatly increased, so that opening does not occur with the greatest pressures the subject can create by assuming the prone position and by flexing the neck toward the chest.

The tube does not open regularly or to the same extent with each act of swallowing but shows great differences in different persons as well as in the same person at different times.

Unless quantitative studies are made, the pathologically patent tube is likely to be overlooked.

When the tube opens to pressures below 20 mm. of mercury it may be considered pathologically patent. The patient experiences crackling sounds and a sense of fulness in the ear even on gently blowing the nose. When extreme patency exists even the small pressures developed in respiration and phonation are sufficient to open the tube.

The tube may then remain continuously open for various periods, from a few minutes to many hours. This permits the direct transmission of voice and breath sounds to the middle ear from the nasopharynx (autophony), masking the hearing for all sounds near threshold entering along the external auditory canal. During quiet respiration the hearing for sounds of about 500 cycles principally is affected.

With the eustachian tube open the changes in pressure during respiration (about 5 mm. of mercury) will cause a flaccid drum or atrophic scars in the drum to move outward on expiration and inward on inspiration, but when the drum is normal it does not move even with deep respiration. Then the auscultating tube may be the only means of obtaining objective evidence of the patent tube. Through it one can hear the distinct blowing sounds of respiration and the reverberation of nasally produced sounds, such as "M" or "N," and when the tube is temporarily closed determination of its patency by the method described reveals the abnormally low pressures required to open it.

The patient can hear sounds delivered to the nasal fossae more readily when the tubes are open than when they are closed. The acoustic energy entering from the nose to the middle ear through a patent eustachian tube is greatest from the ipsilateral nasal fossa.

A large number of factors, found to result in closure or in opening of a pathologically patent tube, are listed in table 1 and afford an insight into the way in which the normal tube reacts to various common stimuli.

Heretofore the cause for pathologic patency of the tube has not been apparent except in rare instances when loss of tissue occurs at the tube, as by ulcerations, and possibly in some cases of marked loss of weight, as from debilitating disease (Ostmann's theory of atrophy of fat about the tube). The study of a group of cases here presented, in which the cause is more clear, indicates that a lack of tonus of the muscles that affect the eustachian tube (tensor veli palatini) and are supplied by the fifth nerve is the principal factor in producing the clinical entity.

The absence of noticeable disturbances in palatal movement in the group of patients operated on, with motor paralysis of the fifth nerve and abnormal patency of the eustachian tube on the same side, indicates that the muscle designated the tensor veli palatini is more properly to be considered a dilator of the tube.

An experimental method for obtaining objective evidence of "autophony" is described, and records so obtained are illustrated.

An experimental method is described which permits investigation of the relations of the duration, extent and time of opening of the eustachian tube during the act of swallowing and their integration with the latter. One of the records obtained in this manner is illustrated.

The eustachian tube is a sensitive neuromuscular mechanism which not only protects the delicate structures of the middle ear from the relatively large changes in pressure normally occurring in the nasopharynx but permits equalization of small differences in pressure normally developing between the middle ear and the world outside.

RHINOSPORIDIOSIS (SEEBER)

FIRST OCCURRENCE IN A FEMALE IN NORTH AMERICA

JACOB RUCHMAN, M.D.
BROOKLYN

Ashworth [1] furnished the first satisfactory description of Rhinosporidium seeberi. It was first mentioned by Seeber,[2] of Argentina, in 1900, and it was independently discovered in India, where the first case of involvement was reported by O'Kinealy.[3] Minchin and Fantham [4] studied O'Kinealy's material and created the name Rhinosporidium kinealyi, which remained in vogue until Ashworth demonstrated Seeber's priority and amended the name to its present form, Rhinosporidium seeberi.

Wright [5] reported the first case of rhinosporidiosis in North America in 1907. The patient was a man aged 20, in Memphis, Tenn. In March 1897 he first came to the attention of Dr. Wright, who removed with a snare a papillomatous-looking growth which sprang from the lower anterior part of the right side of the septum. In November 1898 Dr. Wright removed a smaller growth of similar appearance from the anterior end of the right inferior turbinate body. The septum was healthy. In 1902 the patient had further obstruction on the same side, springing from the old site in the septum. Dr. Wright removed this and for the first time sent a specimen to a pathologist, Dr. W. Krauss. The pathologist sent for Dr. Wright and asked him to look at the slide.

1. Ashworth, J. H.: On Rhinosporidium Seeberi (Wernecke, 1903), with Special Reference to Its Sporulation and Affinities, Tr. Roy. Soc. Edinburgh **53**:301-342, 1923.

2. Seeber, G. R.: Un nuevo esporozoario parásito del hombre dos casos encontrados en pólipos nasales, Thesis, Universidad Nacional de Buenos Aires, 1900; Rhinosporidium kinealyi et Rhinosporidium seeberi, une question de priorité, Ciencia med., 1912.

3. O'Kinealy, F.: Localised Psorospermosis of the Mucous Membrane of the Septum Nasi, Proc. Laryng. Soc., London **10**:109-112, 1903; J. Laryng. **18**: 375-378, 1903; Drawings of Localised Psorospermosis of the Mucous Membrane of the Septum Nasi, ibid. **19**:93-94, 1904.

4. Minchin, E. A., and Fantham, H. B.: Rhinosporidium Kinealyi, n. g., n. sp., a New Sporozoon from the Mucous Membrane of the Septum Nasi of Man, Quart. J. Micr. Sc. **49**:521-532, 1905.

5. Wright, J.: A Nasal Sporozoon (Rhinosporidium Kinealyi), New York M. J. **85**:1149-1152, 1907.

Neither physician knew what the organism was. At about this time Dr. O'Kinealy, of London, England, reported his case and Dr. Krauss, the pathologist, compared notes, slides and specimens, with the resultant diagnosis of the protozoan disease.

The first case in Ceylon was reported by Castellani and Chalmers [6] in 1913. The first European case was reported by Orlandi [7] in 1926. The first case in India was reported by Noronha [8] in 1933.

To date there have been 53 cases reported, from the following locations: 4 from Ceylon, 40 from India, 5 from North America, 3 from South America and 1 from Italy. In 4 of the 53 cases the disease occurred in females. It usually attacks young subjects—in most of the reported cases the patients were under 30. Nothing definite is known about the mode of infection and transmission. The presence of infection in neighboring sites would point to the possibility of autoinoculation. Some authors have expressed the belief that the organism is transmitted in dust or water. The disease may also occur in animals. In view of the rarity of recognized rhinosporidial infection, wide geographic distribution is of extreme interest. There seems no reason to doubt that the organism reported from different parts of the world is a single form. Unrecognized infection has undoubtedly occurred, but even with this possibility in mind, it is evident that successful inoculation of man must be characterized by a remarkable discontinuity.

In 40 of the 53 cases the lesion was in the nose, mainly in the anterior part. The conjunctiva was affected in 8 cases. The ear also has been involved. Beattie,[9] professor of pathology at the University of Sheffield, England, was the first to describe the parasite in the ear. He observed a polypus of the nose which proved to be rhinosporidiosis. In the district of India from which the aforementioned specimen came he had the opportunity to examine some oral polypi, in which he found the parasites. He advised that the name rhinosporidium be abandoned. He devised a staining method for this parasite, which is used to this day.

---

6. Castellani, A., and Chalmers, A. J.: Manual of Tropical Medicine, ed. 3, New York, William Wood & Company, 1919.

7. Orlandi, N.: Granulom der Conjunctiva durch "Rhinosporidium seeberi," Virchows Arch. f. path. Anat. **262**:314-327, 1926.

8. Noronha, A. J.: (*a*) Preliminary Note on the Prevalence of Rhinosporidiosis Among Sand-Workers in Poona, with a Brief Description of Some Histological Features of the Rhinosporidial Polypus, J. Trop. Med. **36**:115-120 (April 15) 1933; (*b*) Further Observations on Rhinosporidiosis, ibid. **36**:368-370 (Dec. 1) 1933.

9. Beattie, J. M.: Rhinosporidium Kinealyi, J. Path. & Bact. **11**:270-275, 1906; A Sporozoon in Aural Polypi, Brit. M. J. **2**:1402-1403 (Nov. 16) 1907.

The staining method is as follows:

1. Stain by Richard Murs alcoholic eosin method, omitting methylene blue.
2. Place in a 2 per cent iron alum solution for fifteen minutes.
3. Transfer to a 0.5 per cent watery solution of hematoxylin for fifteen minutes.
4. Wash and decolorize carefully after the manner of Heidenhain.
5. Stain for about one minute with 0.5 per cent alcoholic solution of *Lichtgrün* (Grühber) and mount in Canada balsam after dehydrating and cleaning.

Recently Pasternack and Alexander [10] reported the sixth case of infection by Rhinosporidium seeberi in this country. On Nov. 2, 1936, a white boy 14 years old came to their attention for treatment of a growth in the nose. The growth completely occluded the left nasal cavity and bled on manipulation. It was removed with a snare. Histologically it proved to be a rhinosporidial polyp. In 1937 there was a recurrence at the same site. It bled freely when touched with the probe. It was excised, and the raw edges were electrically cauterized.

On May 14, 1938, Caldwell and J. D. Roberts [11] reported a case of rhinosporidiosis in the United States. The patient was a white boy aged 16, from Kilgore, Texas. He complained of bleeding from the right side of the nose for three or four months. He had been aware of a mass there for two months. Examination disclosed a coarsely lobulated polypoid mass attached to the anterior superior part of the right side of the nasal septum. The tumor was a reddish gray mass measuring 1.3 by 0.7 by 0.4 cm. Histologic examination disclosed the parasite.

In the nose the characteristic clinical finding is a bleeding polyp, which tends to become obstructive, and for this symptom advice is often sought. Often well marked coryza precedes and accompanies the obstruction. In the discharge, which is thin and mucoid and often stained with blood, the spores and sporangia are usually present. Purulent discharge is rare. If the growth occurs in the nasopharynx, it may hang downward and cause difficulty in swallowing. There is a tendency for the condition to recur, and frequent operations are usually performed. There is often a history of long standing, the infection remaining localized to the original site, and there is no tendency for invasion of the blood stream. The commonest site of the lesion in the nose is the anterior part. The growth is oval or rounded, lobulated and pedunculated and attached by a definite stalk to the mucous membrane. Sometimes the growth arises farther back in the nose and in the nasopharynx. It is

10. Pasternack, J. G., and Alexander, C. S.: Rhinosporidium Seeberi: An Etiologic Agent in the Production of Nasal Polyps, Arch. Otolaryng. **27**:746-765 (June) 1938.

11. Caldwell, G. T., and Roberts, J. D.: Rhinosporidiosis in the United States: Report of a Case Originating in Texas, J. A. M. A. **110**:1641-1644 (May 14) 1938.

usually single but may be multiple. As a rule it takes the form of small nodules or leaflike processes with rounded or dentate margins. The color is pink or red, and papillary projections on the surface give it an appearance that has been likened to that of a raspberry or a strawberry. When the growths are multiple and in the form of tiny nodules, a type sometimes seen in the conjunctiva, the resemblance to granulation tissue is close. The redness is partly due to the vascularity but especially to the free blood so often found on the surface, where there are much adherent mucus and mature sporangia. The involvement varies from granulations the size of a pinpoint to large solitary masses. It is usually friable, soft and vascular and tends to bleed readily on removal.

STRUCTURE AND LIFE HISTORY OF RHINOSPORIDIUM

The organism is a round or oval cell somewhat smaller than a red blood cell, with clear vacuolated protoplasm enclosed by a well defined membrane. The nuclei begin to divide but not the cytoplasm. The division continues until the formation of about 16,000 young spores enveloped in a sporangium about 150 microns in diameter. When the sporangium reaches the size of 200 to 300 microns, it ruptures and the spores escape with some mucoid material into the lymphatic current and enter the connective tissue spaces, and the trophic stage sets in.

HISTOLOGY

Covered by stratified squamous epithelium but in growths from the posterior part of the nasal cavity, the epithelium is columnar and often ciliated. Between papillary processes spores are usually found. The core of the growth consists of vascular fibromyxomatous connective tissue, in which parasites in various stages of development are present. A great many polymorphonuclear leukocytes and red blood cells are often seen surrounding the mass of spores. Giant cells and eosinophils may be present.

The growth is a papillomatous tumor with stroma denser than that of the usual nasal polypus. There is a suggestion of a central supporting framework, free of parasites, and of a looser surface zone of varying depth, in which many parasites are present. There is a generalized low grade inflammatory infiltration, together with frequent small areas of scarring. The parasites are found within the stroma. As the organism enlarges, the cytoplasm becomes heavily granular, and when a diameter of 40 to 50 microns is reached, nuclear division begins. The daughter nuclei repeatedly subdivide, while the entire cyst continues to increase in size. Cytoplasm division finally occurs when the diameter of the cyst reaches 120 to 140 microns, and there results a sporangium filled with a great number of spores. The sporangium expands and dis-

tends to a final diameter of 250 to 300 microns. The spores burst through the thickened membrane and come to the surface of the polyp and finally appear in the nasal discharge.

REPORT OF FIRST CASE OF RHINOSPORIDIOSIS (SEEBER) IN A FEMALE IN NORTH AMERICA

My case is the fifty-sixth to be reported, the eighth case reported in North America and the first on record in North America in which the condition occurred in a female.

The patient was 33 years of age and married; she had 5 children. She was born in Galicia, then a part of Austria.

Her past history revealed nothing that might have any bearing on her present condition. At the age of 10 years she had typhoid fever, from which she recovered rapidly, and with no sequela. She had had no operations.

In her early childhood and adolescent stage, she did not come in close contact with any animals, nor was her mode of living any different from that of her four brothers and three sisters, who had always enjoyed good health. Her father was living and well. Her mother had died of lobar pneumonia. There was no family history of allergy.

She had come to the United States in 1922, at the age of 16, and got a job in a window shade factory. One year later she was married. She settled with her husband in the Bronx, N. Y., lived there for two years and then came to Brooklyn, where her husband opened a butcher shop. From time to time she helped her husband in his trade, even at cutting and dressing the meats.

For many years she had apparently enjoyed good health. She was never troubled with head colds, sinus disease or infections of the upper part of the respiratory tract until two and a half years before she came to my attention, when the current trouble began. She claimed that she had caught cold and the left side of her nose became stuffed. This nasal blocking became worse with time, but since she had no other symptoms she did not seek medical aid. Some time later, she noticed that when she blew her nose her handkerchief was stained with blood, and that it came from the left side of her nose. This continued for two years, until six months before examination, when she began to suffer from headaches. These headaches were limited to the left side of her head and radiated toward the occiput, left ear and back of the neck. The pains became aggravated as time went on, to such an extent that they interfered with her sleep. They became constant, and even drugs of the more potent type soon lost their soothing powers. It was for this agonizing symptom that she began to seek medical relief. On Oct. 28, 1937, she was referred to me by her family physician.

The patient appeared tired and worn out from lack of sleep. Deep lines were visible on her face, and the drawing of the muscles on the left side of the face almost to closure of the left eye denoted the terrific pain she was undergoing.

After taking a detailed history I prepared her for an examination. Transillumination of her sinuses showed her left antrum to be darker than her right, but not sufficiently dark to indicate the presence of pus. Examination of the nose revealed the following facts: The right side, except for a slight mucous discharge, appeared normal. The left side showed a partial closure between the septal wall and the inferior turbinate body. At first it appeared like a septal-turbinal synechia, but after application of 10 per cent cocaine and epinephrine hydrochloride the inferior turbinate shrank and left the septal wall. Attached to the lower anterior part of

the septum was an elevated growth the size of a pea, leaflike, and with well defined points radiating in all directions. The surface was smooth and the color pale red. On slight manipulation the growth began to bleed. A mucopurulent discharge stained with fresh blood was spread over the inferior turbinate and the floor of the nose. After suction of all this discharge I noticed a large red polypous mass right underneath the middle turbinate. On manipulation it bled profusely. I had never seen such a picture before. Posterior rhinoscopic examination did not reveal anything abnormal. The throat and ears were normal. A roentgenogram of the sinuses showed cloudiness of the ethmoid sinus and the antrum, but their outlines were distinct and intact. There was no suggestion of fluid.

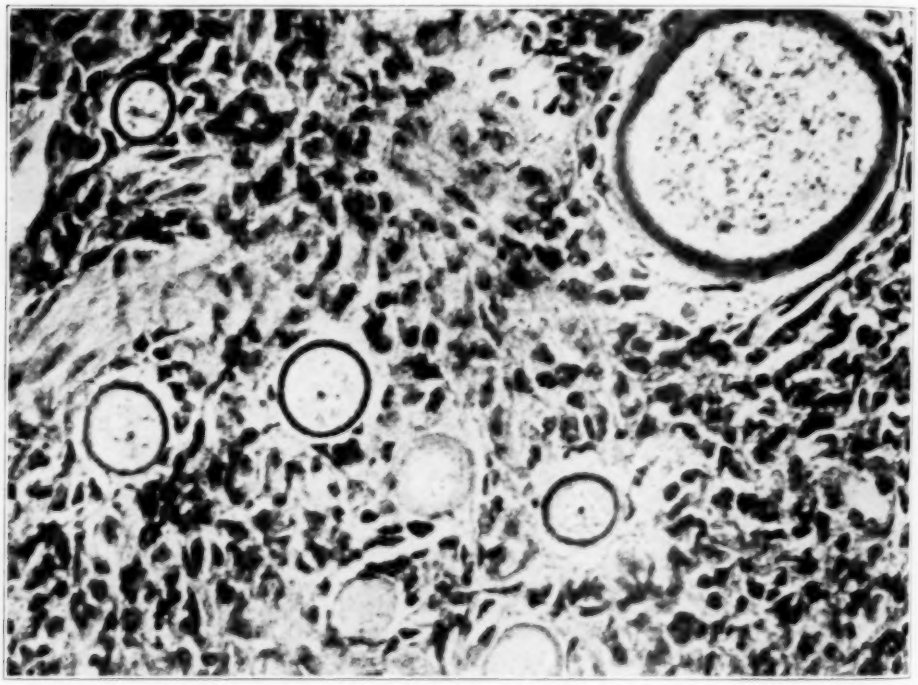


Fig. 1.—Growth of a sporangium; × 500.

On Nov. 19, 1937, I took a biopsy specimen from the polypous mass and one from the growth of the septum and sent them for examination to Dr. A. Plaut, pathologist at the Beth Israel Hospital, New York. To my great surprise he made a report of rhinosporidiosis (Seeber). Dr. Plaut's enthusiasm was greatly aroused. He had never seen the organism before but remembered reading about it ten years before. He quickly made comparisons, and the diagnosis followed.

I then proceeded with the removal of the septal growth and the polypous mass, which was embedded in the middle meatus and attached to the middle turbinate body. In order to make a complete removal, I had to remove part of the middle turbinate. Bleeding was profuse. Repeated packing was necessary. When the bleeding was controlled some of the leaflike processes of the original mass were found to extend over the ethmoid area. I was curious to know whether the

ethmoid cells were involved, and in view of the roentgenogram of the left side, I opened the left anterior ethmoid cells and found them in a normal, healthy condition. I also punctured the left antrum, and after irrigation washings returned clear.

On December 8 and 18 there were regrowths at the original sites; they were removed with a cold snare and forceps and sent to the laboratory. Again there was profuse bleeding.

At the suggestion of Dr. Wright,[12] I prescribed treatment with antimony and potassium tartrate, 5 per cent aqueous solution, in the nose, followed by application of chlorhydroxyquinoline ointment, with the hope that there might be no further recurrence of the growth.

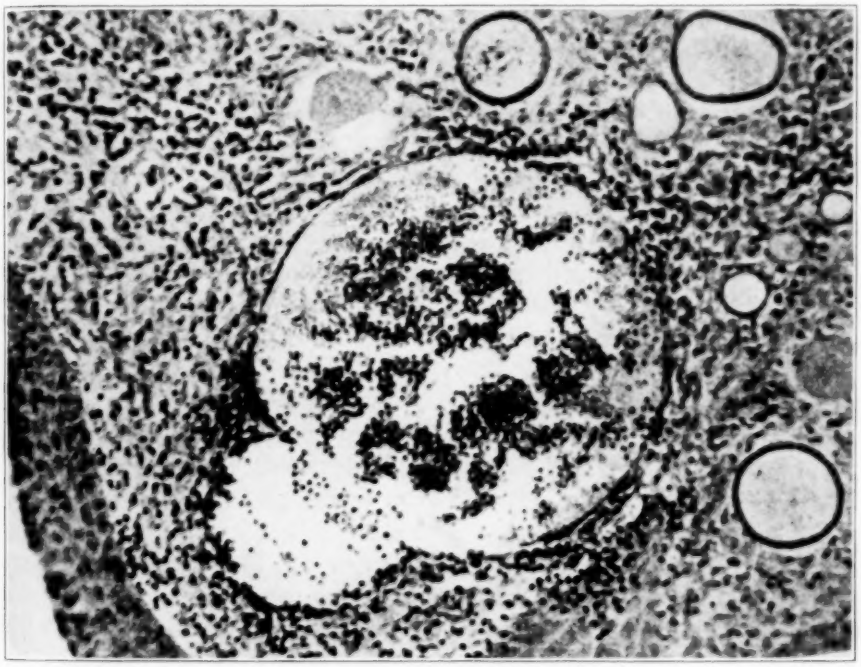


Fig. 2.—Rupture of a sporangium discharging spores; × 700. Note the immature spores surrounding the sporangium.

On Jan. 8, 1938, the patient showed a marked improvement both clinically and physically. Her headaches had subsided greatly. The tired, drawn expression of her face was gone. She was more cheerful and optimistic. The sites of the original infection of the nose revealed a remarkable change. The septal wall had a normal healthy appearance. It was separated from the turbinate. There

12. Wright, R. E.: Rhinosporidium Kinealyi of the Conjunctiva, Indian M. Gaz. **57**:6-7 (Jan.) 1922; Rhinosporidium Kinealyi of the Conjunctiva Cured by Tartrated Antimony (Tartar Emetic), and Notes on a Case in Which the Lachrymal Sac Was Affected by This Sporozoon, ibid. **57**:81-82 (March) 1922; Rhinosporidium Kinealyi of the Conjunctiva, ibid. **57**:82-83 (March) 1922.

were no evidences of further growths. The lateral wall of the nose showed a similar picture. There were no further signs of polypoid masses. No bleeding occurred on manipulation. The treatment was continued until Feb. 1, 1938, at which time all clinical and physical evidences of the disease had disappeared. After six months of complete rest she was again observed. The nose was normal in all respects. She did not experience any nasal bleeding and once more enjoyed normal health.

SUMMARY AND CONCLUSIONS

I present a case of rhinosporidiosis because of its rarity, this being the first reported case of the disease in a female in North America.

Rhinosporidiosis is a fungous infection involving the mucous membrane of the nose, ear, conjunctiva, pharynx, larynx and penis. The disease has been reported mostly from India, Ceylon, Africa and the Philippine Islands and occasionally from North and South America. The cause of the original infection is unknown. I cannot explain how autoinoculations at new sites take place.

Characteristically a polypous mass appears, with profuse bleeding. The manifestations are local, with no invasion of the depths of the surrounding areas or the blood stream. The spores are mixed in the nasal secretions and discharged from the nose. Recurrences are rapid.

Experimental transplantations into animals have not succeeded.

The characteristic picture under the microscope establishes the diagnosis.

Repeated operations for the removal of the growth and its adjacent normal tissue are often necessary. The treatment may be supplemented by the use of chlorhydroxyquinoline ointment and of a 5 per cent aqueous solution of antimony and potassium tartrate.

NASAL ALLERGY IN CHILDREN

E. J. BARNETT, M.D.
AND
H. D. CARNAHAN, M.D.
SPOKANE, WASH.

That allergy is a common cause of rhinitis in children, whether acute or chronic, seasonal or perennial, recurrent or constant, is often unrecognized.[1] The majority of adults with rhinitis date their disturbance from the first decade of life. It is reported that 44 per cent of patients with chronic nasal complaints seen in routine otolaryngologic practice have nasal allergy.[2]

We, as pediatricians, are not hesitant to present the problem of nasal allergy of childhood before rhinologists, because this constitutional disturbance is the mutual concern of every rhinologist and every physician who treats the sick child. Nasal allergy is not a local disease, to be treated by local measures only.[3]

No doubt much of the present criticism of rhinologists by patients with chronic nasal complaints who have been treated unsuccessfully by local applications or operations is justifiable. Too often either the associated nasal allergy has not been recognized or coordinated attack along allergic lines has not been instituted. Because of the persistence of unrelieved allergy or the occurrence of associated infection, not only may the nasal condition become more confused and complicated, but chronic bronchial asthma may be superimposed.

SYMPTOMS

With the commonly observed recurrent type of allergic rhinitis in children, the parents complain that the child "has one cold in the nose after another." It is their observation that the "cold" lasts not for a few days only but even for weeks. Also, after but a few days of relief, and for no apparent reason, the "cold" may suddenly recur.

Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Spokane, Wash., Jan. 29, 1939.

1. American Academy of Pediatrics: Panel Discussion on the Role of Allergy in Pediatric Practice, J. Pediat. **13**:582 (Oct.) 1938.

2. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses, St. Louis, C. V. Mosby Company, 1936.

3. Tuft, L.: Clinical Allergy, Philadelphia, W. B. Saunders Company, 1937.

Even in the intervals between "colds," the nose is stuffy, especially in the morning.

Sneezing, sniffling and nasal itching, obstruction and discharge are characteristic. Epistaxis occurs frequently. The eyes may be red and itching; the speech is often nasal. Fever is not observed unless secondary infection complicates the allergy, but great irritability is usually observed in conjunction with these manifestations.

Difficulty in nasal breathing or lack of it tires the child and results in broken sleep. Insomnia may be aggravated by overdosage of ephedrine. The resulting fatigue is often a pronounced symptom, but it is insufficient to require rest in bed. Too often the children are needlessly confined to bed for relief of their afebrile, noninfectious "colds" or for protection of their associates. Other members of the family, playmates or schoolmates do not become secondarily affected. In bed or confined indoors the allergic child may be exposed to an aggravated atmosphere of the very inhalants to which he is sensitive.

If tonsillectomy and adenoidectomy have been performed for relief of such "colds" the parents often emphatically state that not only has relief not been obtained but the "colds" are even intensified. Mouth breathing is frequently so manifest that the parents are persuaded the adenoids have recurred.

HISTOPATHOLOGIC CHANGES [2]

The gross changes in the nose are the result of edema and eosinophilic infiltration of the tissues. The nasal mucous membrane is characteristically pale and edematous. The secretion is usually serous or mucoid. Polyps may be associated with the condition but often disappear after control of the allergic factor. The membrane does not usually become red and thickened, and the secretion does not become purulent unless secondary infection develops. Edematous or cystic changes in the paranasal sinuses may be associated with the condition.

The properly stained nasal secretion shows many and predominating eosinophils. In contrast, a predominance of neutrophils indicates an associated infection, which at times may be so severe that eosinophils are not found until it is disappearing.

DIAGNOSIS [2]

Routine smears should be examined to arrive at accurate diagnosis of nasal conditions. The demonstration of eosinophils corroborates the diagnosis of nasal allergy.

The technic is simple and should be carried out repeatedly in the office of every rhinologist and every physician who cares for a child

with nasal disturbances. Granting that the history of familial allergy, the nasal symptoms of the child, the occurrence of serous or mucoid nasal discharge, the appearance of the nasal mucous membrane and the presence of other allergic manifestations, such as asthma or eczema, are most significant in diagnosis, cytologic examinations of the nasal secretions also are important and necessary. The diagnosis may be regarded as confirmed if allergic measures afford relief.

It is our routine to make a cytologic examination of the nasal secretions each time a child with nasal symptoms is presented. We feel strongly that every rhinologist's office should likewise be equipped for carrying out this important examination as a routine.

It seems evident from the cytologic examinations that allergy can be diagnosed when eosinophils predominate. With both eosinophils and neutrophils in large numbers the condition is undoubtedly allergy plus infection. A return of eosinophils alone suggests that the infection has disappeared. With neutrophils only the nasal disturbance is either infection alone or infection severe enough to overshadow allergy completely. In the latter contingency only repeated cytologic examinations with final appearance of eosinophils can make the direct diagnosis possible. With repeated failure to find eosinophils definite evidence of active allergy is absent.

We find that routine cytologic examination of nasal secretions is highly informative and gives tremendous personal satisfaction. The parents are more than appreciative of the efforts made in attempting to determine whether each successive or every persistent "cold in the nose" in their child is allergic or infectious. Moreover, they not only readily accept such a laboratory diagnosis of allergy but are more than willing to carry out prophylactic allergic measures in the home.

We appreciate that the presence of eosinophils is a guide only and that a negative smear does not exclude allergy. In cases in which allergy is strongly suspected we prefer to repeat the examinations of the nasal secretions and to delay strict prophylactic allergic measures until at least one smear is frankly positive for eosinophils.

CYTOLOGIC EXAMINATION

Using a cotton swab, one should obtain secretion from each nostril separately and spread it thinly on two cover slides. In the allergic child sneezing will often occur at the time of the swabbing. With fresh Giemsa stain the granules of eosinophils will appear brownish red and coarsely stained. The slide should be examined with an oil immersion lens.

Dr. George Piness and Dr. Hyman Miller supplied this modification of the staining technic. The slides are fixed in formaldehyde vapor by

standing upright for at least two hours but preferably overnight in a covered glass jar. Cotton in the bottom of the jar is kept moistened with solution of formaldehyde. The slides are then rinsed with dehydrated methyl alcohol. To 5 cc. of distilled water in a test tube 1 drop of 1 per cent acetic acid and 5 drops of Giemsa stain are added, and the mixture is shaken gently but well. This stain is poured on the slide and left for one-half hour. The slide is then washed with distilled water and blotted dry.

PROPHYLACTIC MEASURES

In an allergic child hypersensitiveness to inhalants, contactants, pollens and foods is the rule. The child's allergy is rarely due to a single cause; multiple and mixed factors are involved.

It is gratifying to observe the relief that can be obtained by many children afflicted even with severe nasal allergy through the use of moderate and practical prophylactic measures. Local nasal treatment is often unnecessary. In fact, results at times seem enhanced if all nasal treatments are dispensed with.

To avoid specific animal emanations the house must be thoroughly cleaned after all pets are prohibited indoors. Feather pillows should be covered with cellophane slips or the feathers replaced by a good grade of cotton entirely free from cottonseed. The mattress should be enclosed with rubberized sheeting. A plain, bare, dust-free sleeping room, with surfaces waxed or wiped with damp or oiled cloths, is indicated, and during the sweeping or dusting of the house the child is to be kept out of doors. Contact with insecticides or sprays must also be avoided, and the use of unscented soaps and nonallergic cosmetics by the entire family is highly advisable, as is also avoidance of excessive smoking in the child's presence. These measures are all sound precautions against allergens or irritations.

Allergic colds are more frequent and more persistent in the winter, not only because of greater confinement, which increases contact with offending inhalants in the house, but also because of excessive exposure to wool in the heavier bedding, clothing, sleeping bags and night garments. The practice of permitting allergic children to sleep in the winter between woolen blankets instead of sheets is to be condemned. So also is the presence of woolly toys. It is usually possible for the allergic child to sleep in his cotton summer night clothing if sufficient warmth is provided from heavier blankets enclosed on both sides by sheets. To avoid "physical" allergy from the cold winter night air, the child's bedroom window should remain closed. Fresh air, with the chill off, may be obtained from another part of the house.

In childhood, hypersensitiveness to foods may be overcome. On the other hand, the allergic child may acquire sensitivity to foods

formerly harmless that he takes in excess. Nuts and chocolate in all forms are apparently so often offenders that we routinely stop their allowance in the diet. Also, it is well to replace fresh milk by unsweetened evaporated milk. For at least a few weeks, consumption of eggs, fish, wheat, oranges and prepared lards and salad dressings containing cottonseed oil may be discontinued. Ordinarily the allergic child receives excessive amounts of cod liver oil without relief of his colds. Hence, in practice, cod liver oil often needs to be withdrawn entirely in order to rule out sensitivity to it. With relief of symptoms, withdrawn foods may be restored, one at a time, in the attempt to determine specific sensitivity.

Removal of adenoids, when necessary, is probably not advisable for the allergic child until after the frosts, in order to avoid institution or aggravation of hay fever due to pollen.

ALLERGIC STUDY AND HYPOSENSITIZATION

It is our usual practice to carry out allergic skin testing and hyposensitization only when prophylactic measures have not given sufficient relief from marked or complicated conditions. In this we differ from allergists who believe that every allergic child should be tested because of the possibility of later aggravation or complication of its allergy. We are also reluctant in most instances, even though we recognize the typical nasal appearances of allergy, to institute testing without a preliminary examination by a rhinologist, to rule out complicating nasal conditions or those requiring surgical treatment. Then when the allergic child is tested he is tested thoroughly.

Over fifty scratch tests, with 1:50 dilutions, are made, with pollens, with all epidermal substances and with such foods as eggs, milk, wheat, chocolate, nuts and cottonseed, to all of which children may be alarmingly sensitive. Using about two hundred allergens in 1:1,000 dilution, we then test intradermally. Most authorities believe that intradermal testing is more accurate than, and far superior to, scratch tests. To our surprise, the intradermal testing does not disturb the child to any greater degree than do the scratch tests.

Foods that produce a reaction are entirely removed from the diet. Later, the less reactive foods first are permitted, with slow and cautious addition of those to which more sensitivity is shown as symptoms are further relieved.

Hyposensitization, with a compound extract of offending allergens, is carried out by injections, beginning as a rule with a 1:50,000 dilution. Subsequent dosage and spacing of injections follow no set rule but are changed or modified according to individual indications.

COMMENT

Both rhinologists and pediatrists must become allergic minded to arrive at accurate diagnosis and to obtain better results in the management and treatment of many nasal complaints in children.

To determine nasal allergy in childhood and to determine whether or not the allergy is complicated by infection, physicians need to perform routine examinations of the nasal secretions. The value of such laboratory routine cannot be overemphasized. It is well to note that the volume of the smears in this routine practice is too great to permit dependence on a clinical laboratory. Moreover, the tests are of more clinical value if carried out in the physician's office, where the results may be closely compared to the progress of the patient.

Allergic control is vital if necessary associated surgical procedures are to be of benefit. Removal of infected tonsils and adenoids may aggravate symptoms in a child with uncontrolled allergy. Disappointment and discouragement will follow nasal treatments and operations on the sinuses or nose, even though the procedure is indicated by roentgen or clinical findings, if associated allergy is not simultaneously or previously relieved. Removal of nasal polyps in the patient with uncontrolled allergy may be followed by prompt recurrence.

CONCLUSIONS

1. Allergy should always be suspected as the cause of rhinitis in children.

2. Cytologic examinations of the nasal secretions with predominance of eosinophils offer confirmatory evidence.

3. Routine repeated cytologic examinations for all patients with chronic nasal symptoms are important.

4. Local treatment for nasal allergy must be conservative.

5. Disappointments in rhinologic practice often result from failure to recognize and relieve associated allergy.

6. Prophylactic allergic measures are often sufficient to give relief from nasal allergy.

7. Allergic testing and hyposensitization may be reserved for unrelieved or complicated conditions.

8. The importance of allergy to the rhinologist cannot be overemphasized.

OTITIC SINUS THROMBOSIS CAUSING INTRACRANIAL HYPERTENSION

W. JAMES GARDNER, M.D.
CLEVELAND

In this presentation an explanation is sought for the syndrome of increased intracranial pressure which occasionally accompanies an attack of otitis media and which is not due to meningitis, encephalitis or abscess of the brain.

Quincke [1] in 1896 described a syndrome in which the signs of increased intracranial pressure were associated with the finding of clear cerebrospinal fluid on lumbar puncture. He stated the belief that in such cases an excess of cerebrospinal fluid results from hypersecretion of the choroid plexus. He referred to the condition as "serous meningitis" and described otitis as one of the predisposing factors. Various terms have since been coined to describe and label conditions of this type, such as "cerebral pseudoabscess," "meningismus," "serous meningitis," "serous ependymitis," "arachnoiditis," "hypertensive meningeal hydrops," "toxic hydrocephalus," and "otitic hydrocephalus." Recent articles by Symonds,[2] Frazier,[3] Dandy,[4] Davidoff and Dyke,[5] McAlpine,[6] Williams,[7] and Ersner and Myers [8] described the authors' experiences with these conditions and offered suggestions as to the probable cause or causes. Some of the authors did not attempt to separate the otitic from the nonotitic form. The literature on this subject has been so well discussed in some of these articles that it will not be reviewed here.

From the Cleveland Clinic Foundation.

1. Quincke, H.: Ueber Meningitis serosa und verwandte Zustände, Deutsche Ztschr. f. Nervenh. **9**:149-168, 1896-1897.

2. Symonds, C. P.: Hydrocephalic and Focal Cerebral Symptoms in Relation to Thrombophlebitis of the Dural Sinuses and Cerebral Veins, Brain **60**:531-550 (Dec.) 1937.

3. Frazier, C. H.: Cerebral Pseudotumors, Arch. Neurol. & Psychiat. **24**:1117-1132 (Dec.) 1930.

4. Dandy, W. E.: Intracranial Pressure Without Brain Tumor: Diagnosis and Treatment, Ann. Surg. **106**:492-513 (Oct.) 1937.

5. Davidoff, L. M., and Dyke, C. G.: Hypertensive Meningeal Hydrops, Am. J. Ophth. **20**:908-927 (Sept.) 1937.

6. McAlpine, D.: Toxic Hydrocephalus, Brain **60**:180-203 (June) 1937.

7. Williams, H. L.: Otitic Hydrocephalus, Arch. Otolaryng. **25**:632-652 (June) 1937.

8. Ersner, M. S., and Myers, D.: Otitic Hydrocephalus, with a Suggestion as to Etiology, Ann. Otol., Rhin. & Laryng. **45**:552-566 (June) 1936.

Interest in the problem was aroused by an article by Symonds, which appeared in 1931.[9] He applied the term "otitic hydrocephalus" to the syndrome in which infection of the ear is followed by increased intracranial pressure, without definite localizing signs, evidenced by choking of the optic disks and increased spinal fluid pressure (but no increase in the cell count), which clears up after repeated spinal or ventricular punctures. Without encephalographic proof, he assumed that hydrocephalus was present. In a more recent article,[2] he concluded that in this syndrome infection of the lateral sinus is the most important etiologic factor but that *obstruction* of the sinus is not essential. He suggested that mural thrombophlebitis of the superior longitudinal sinus by retrograde extension from the lateral sinus may, by putting out of action a sufficiently large proportion of the arachnoid villi, so interfere with absorption of cerebrospinal fluid as to cause "otitic hydrocephalus." From a study of my own cases, I also had arrived at the conclusion that the syndrome is due to sinus thrombosis. However, the encephalographic studies which were carried out will not permit a diagnosis of hydrocephalus. It appears to me that the increased intracranial pressure is due not to hydrocephalus but to intracranial venous engorgement occasioned by interference with the outflow of venous blood from the cranial cavity.

SINUS THROMBOSIS DEMONSTRATED AT AUTOPSY IN TWO CASES

The case in which sinus thrombosis was suggested as the cause of the increased intracranial pressure in this condition was observed in 1934.

CASE 1.—A girl of 9 years gave a history which stated that two years previously a discharge from the left ear had suddenly developed without previous pain. The discharge ceased after a few days, and there appeared to be no sequelae. However, about one month later, while the patient was on a visit to another city, it was found that the vision was seriously impaired by marked choking of the optic disks. Subtemporal decompression was performed on the right; the choking of the optic disks subsided, and the vision returned. The patient had been in good health since that time, but there was constant bulging at the site of the decompression, although this hernia was not tense.

Three days prior to her entry into the hospital, on Jan. 20, 1934, a second attack of otitis media on the left developed. At the time of admission, her temperature was 105 F., and she appeared ill. During the next few days there was marked diurnal fluctuation in the temperature, and on the seventh day of her illness mastoidectomy was performed on the left, disclosing a large amount of pus in the mastoid. A perisinal abscess was present, and it was noted that the sinus was white instead of the usual deep blue. Culture revealed the type III pneumococcus. The temperature continued to fluctuate, and on the twenty-first day of the illness the left internal jugular vein was ligated. The temperature subsided somewhat after this, but the hernia became tense and choking of the optic

9. Symonds, C. P.: Otitic Hydrocephalus, Brain **54**:55-72 (April) 1931.

disks began to appear. The spinal fluid pressure was found to be 400 mm. of water. The fluid contained 110 cells, 60 per cent of which were lymphocytes and 40 per cent polymorphonuclears. After this, definite purulent meningitis developed, and the type III pneumococcus was cultured and the patient died on the thirty-fourth day.

At postmortem examination the left lateral sinus was seen to be represented merely by a fibrous cord, apparently indicating that thrombosis of this sinus had occurred with the attack of symptomless otitis two years previously and that this thrombosis, which had healed without suppuration, had been the cause of increased intracranial pressure at that time. There was acute thrombophlebitis of the left superior petrosal sinus and of the sagittal sinus, together with acute, diffuse, fibrinopurulent leptomeningitis.

In this case, after recovery from "otitic hydrocephalus," the observations at autopsy indicated that the syndrome was produced by thrombosis of the left lateral sinus. This led to the belief that the condition which Symonds had described in 1931 [9] under the title "otitic hydrocephalus" may be due to thrombosis of the intracranial sinuses which, by partially obstructing the outflow of venous blood from the cranial cavity, causes cerebral venous engorgement and a consequent rise in intracranial pressure. Otologists have long been aware that papilledema occurs in a considerable percentage of cases of sinus thrombosis complicating otitis media, but Symonds, in 1937, was the first to offer convincing evidence that the syndrome under discussion is due to sinus thrombosis.

A fact not universally recognized is that sinus thrombosis may be asymptomatic and may occur in the course of mild otitis media. Also, if secondary infection of the thrombus does not occur it will heal without suppuration and usually with recanalization. As recently stated by Sutherland,[10]

A thrombus is essentially a protective measure and is not, as the minority believe, infected from the beginning. It is nature's method of blocking the blood channel in an effort to prevent further spreading of an infection. It is the secondary infection of the clot and not the thrombus that constitutes the danger.

After the experience with the patient whose case has been described, a search of the records disclosed another case, again with autopsy, which clearly demonstrates these facts. In this case also the patient presented the clinical picture which Symonds has called otitic hydrocephalus.

Case 2.—A girl of 7 years was admitted to the hospital on Jan. 13, 1930, the twenty-first day of her illness, because of paroxysms of frontal pain and choking of the optic disks. The illness had begun with pain in the right ear and general malaise. The pain in the ear soon subsided, without discharge. Paroxysms of frontal pain began on the twelfth day, and from the fourteenth to the eighteenth day there was mild fever. On the twenty-first day there was no clinical evidence of mastoiditis and no fever. The patient did not appear ill. There were no abnormal neurologic findings, but there was 4 diopters of choking of the optic disks. Roent-

10. Sutherland, J. M.: Otitic Sinus Thrombosis, Arch. Otolaryng. **27**:1-34 (Jan.) 1938.

genograms of the skull revealed no abnormalities, but those of the mastoids disclosed subacute mastoiditis on the right side. The spinal fluid pressure on the twenty-second day was over 700 mm. of water. The fluid contained 1 cell. The Pandy test gave a negative reaction. On this day a ventriculographic examination was attempted. As the dura was incised, copious fluid escaped, and when the operator introduced a brain cannula the ventricles were not encountered. Mastoidectomy was then performed on the right, and no pus was disclosed. The lateral sinus appeared normal. During the next three days, fluid drained from the incision made in the scalp in the attempt at ventriculographic examination. On the twenty-seventh day the pressure was again over 700 mm. of water, which was the greatest height the manometer would record. The fluid on this occasion contained no cells. On the twenty-eighth day, because of the high intracranial pressure, subtemporal decompression was performed on the left. A copious flow of clear, colorless subdural fluid occurred (estimated at 2 ounces [60 cc.]), after which the intracranial pressure was entirely controlled. After the operation the fluid continued to collect at the operative site, causing marked herniation, and had to be withdrawn daily by means of a needle introduced beneath the temporal muscle. Analysis of this fluid on the forty-sixth day revealed 2 cells per cubic millimeter and a total protein content of 40 mg. The spinal fluid on the same day contained 1 cell and 19 mg. of protein. On the forty-ninth day, encephalographic examination was performed after preliminary injection of dye into the hernial sac. With the patient in the sitting position, 90 cc. of fluid was removed and a similar quantity of air substituted. At the conclusion of the exchange, the spinal canal was entirely empty, and the fluid which had been removed contained none of the dye which had been injected into the hernial sac. This test indicated that the fluid encountered at the decompression was really subdural and not subarachnoid. The resulting encephalograms showed slight dilatation of the left ventricle, with the midline of the brain displaced slightly to the right. There was less subarachnoid air on the left side of the convexity of the brain than on the right. These changes were considered secondary to the subtemporal decompression on the left. On the fifty-sixth day the patient died of pneumococcus type III meningitis of five days' duration.

The significant postmortem observations were as follows: Sections of the superior sagittal sinus, through the anterior, the middle and the posterior third showed pronounced acute suppurative endophlebitis and thrombosis, with marked thickening of the wall of the sinus, proliferation of the intima and organization of thrombus (fig. 1). In the anterior third there was a recent red thrombus, with little suppuration. In the middle and the posterior third, suppuration predominated. Section of the straight and the left lateral sinus showed acute suppurative endophlebitis and thrombosis. Section of the right lateral sinus showed endophlebitis, less acute and with more organization than in the left. Section of the right lateral sinus near the jugular foramen showed organized, canalized thrombosis, with no active cellular infiltration.

In attempting to explain this clinical picture it would seem reasonable to suppose (1) that thrombosis of the right jugular bulb occurred with the onset of the otitis, as the process in this area histologically appeared much older, (2) that at this time a sterile subdural effusion occurred as a result of either the thrombosis or neighboring external pachymeningitis, (3) that this subdural effusion, plus the venous congestion occasioned by the thrombosis of the right jugular bulb, was responsible for the

tremendously high intracranial pressure and (4) that the thrombophlebitis gradually spread from the right jugular bulb to most of the major intracranial sinuses, as well as cerebral veins, with meningitis terminating the picture.

SUBDURAL EFFUSION

Subdural effusion complicating otitis media has not received much mention in medical literature. Penfield,[11] in reporting a case in 1923,

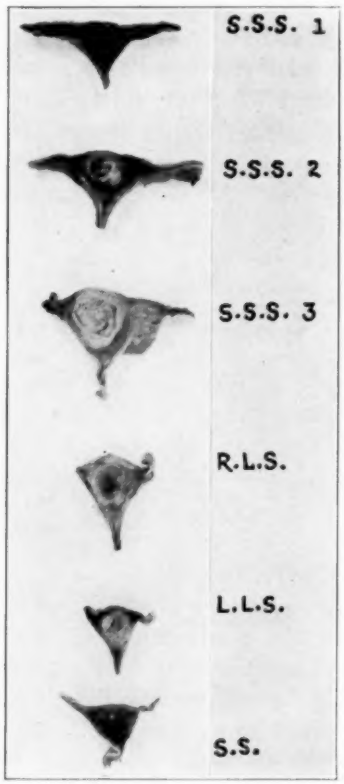


Fig. 1 (case 2).—*S.S.S. 1, 2* and *3,* sections through the superior sagittal sinus in the anterior, the middle and the posterior portion; *R.L.S.,* right lateral sinus; *L.L.S.,* left lateral sinus; *S.S.,* straight sinus.

was the first to describe it. In 1924, by an ingenious process, he[12] demonstrated that a small amount of yellow fluid is present normally in the subdural space of the dog. He concluded, therefore, that the subdural space in man contains normally a small amount of fluid. Patho-

11. Penfield, W.: Subdural Effusion and Internal Hydrocephalus, Am. J. Dis. Child. **26**:383-390 (Oct.) 1923.

12. Penfield, W.: The Cranial Subdural Space, Anat. Rec. **28**:174-175, 1924.

logically this fluid may be augmented by subdural bleeding resulting from trauma or by an effusion secondary to a contiguous area of inflammation. In the cases of otitis under consideration, the subdural fluid is undoubtedly formed as a sterile transudate from the inner surface of the inflamed dura. It may thus be compared to the sterile transudates which occur in other cavities of the body when there is contiguous inflammation. As it accumulates in the subdural space, however, it is not absorbed, as nature has provided no means of absorption from this area. As time goes on it may gradually increase, even after the external pachymeningitis has subsided, because an accession of cerebrospinal fluid is drawn through the arachnoid membrane into the subdural space by the higher osmotic pressure of the subdural fluid due to its higher protein content. That infective sinus thrombosis can act in the same way as external pachymeningitis in producing a sterile subdural effusion is obvious when one considers that the inner surfaces of the dural sinuses form part of the limiting membrane of the subdural space. Since the subdural space continues over the pacchionian bodies and the arachnoid villi, as demonstrated by Winkleman and Fay,[13] it appears likely that subdural effusion would be particularly active if the thrombophlebitis reached the venous channels serving these structures. A subdural effusion, therefore, following an attack of otitis media means that external pachymeningitis or infective sinus thrombosis has occurred.

Subdural effusion should not be confused with nor should it be called external hydrocephalus. The diagnosis depends on the recovery from between the dura and the arachnoid of fluid which contains an amount of protein definitely in excess of that in the subarachnoid fluid. This subdural fluid is generally yellow, and I have observed a protein content up to 700 mg. per hundred cubic centimeters. It is sometimes difficult to demonstrate the presence of subdural fluid when the skull is trephined. This is particularly true if the operation has been preceded by a subarachnoid or ventricular injection of air, because when the cavities of the brain contain air its weight is correspondingly lessened, so that it will float on the subdural fluid, which in turn tends to gravitate to the lowermost portion of the cranial cavity and away from the trephine opening. The presence of subdural fluid can be demonstrated more readily if the patient is operated on in the sitting position and the trephine opening is made in the lower temporal region.

REPORT OF ADDITIONAL CASES

CASE 3.—A boy of 8 years was admitted to the hospital on March 26, 1933, in the eleventh week of his illness, because of pain in the left eye and choking of the optic disks. The history stated that an attack of chickenpox was followed by

13. Winkleman, N. W., and Fay, T.: Pacchionian System: Histologic and Pathologic Changes with Particular Reference to Idiopathic and Symptomatic Convulsive States, Arch. Neurol. & Psychiat. **23**:44-64 (Jan.) 1930.

pain in the left eye and that two weeks later the patient was operated on for mastoiditis on the right. At the operation thrombosis of the lateral sinus was disclosed. The lateral sinus was incised and packed, and the right internal jugular vein was ligated in the neck. The pain in the left eye continued after the operation. There was mild, persistent fever, but no headache, vomiting or diplopia. On admission, because of the general well-being of the patient and the absence of neurologic signs, abscess of the brain was considered improbable. A roentgenogram of the skull showed nothing of significance. Spinal puncture revealed a pressure which fluctuated widely, between 300 and 550 mm. of water. Because of these wide variations in pressure, the Tobey-Ayer test was unsatisfactory. The fluid contained 1 cell; the total protein content was 10 mg. per hundred cubic centimeters; the reaction of the Wassermann and the colloidal gold chloride test were negative. There was choking of the optic disks of 2 diopters in the right and 1.5 diopters in the left eye, and there were numerous hemorrhages on both

Fig. 2 (case 3).—Mild dilatation of the right ventricle with the sulci poorly shown on the right side and absent on the left.

disks. The patient was treated by repeated lumbar punctures. The pressure gradually subsided to normal, and he was discharged after three weeks. After his discharge, however, the spinal pressure increased again to 350 mm. of water, and he was readmitted. Encephalographic examination was performed on April 24, in the fifteenth week of the illness; 112 cc. of spinal fluid was recovered and the encephalograms showed mild dilatation of the right ventricle, with sulci poorly shown on the right side and absent on the left (fig. 2). Spinal punctures were then performed every few days, and by the twentieth week the pressure had gradually subsided to 285 mm. of water. Continued improvement in the appearance of the optic disks then indicated that the pressure was gradually subsiding, and no further punctures were done.

It is interesting to note in this case that, although the total amount of cerebrospinal fluid which was recoverable at encephalographic examination was 112 cc., at subsequent spinal punctures in which the fluid

was not replaced with air the volume removed measured from 50 to 70 cc. This suggests that the increased intracranial pressure was due to cerebral venous engorgement, since, despite the large quantity of fluid obtainable at spinal puncture, there was not an increase in the total volume of cerebrospinal fluid as measured at encephalographic examination. Therefore, the term "otitic hydrocephalus" as applied to these cases is not strictly correct.

CASE 4.—A youth of 18 was admitted to the hospital on August 24, 1937. He had had otitis media on the right seven months previously and mastoidectomy on the right two months later. After the mastoidectomy, he noticed gradual failure of vision, especially in the left eye. He also had occasional headaches on the left

Fig. 3 (case 4).—Normal encephalogram.

side and occasional vomiting. He did not appear at all ill. Aside from a slight weakness of the right angle of the mouth, there were no objective findings. There was choking of the optic disks of 3 diopters in the right eye and 2 diopters in the left eye. The visual acuity was reduced to 6/60 in the right eye, and with the left eye he could barely count fingers. A roentgenogram showed no abnormality in the skull. The spinal fluid pressure was 350 mm. of water. It rose to 400 mm. on compression of the right jugular vein, 450 mm. on compression of the left jugular vein and 500 mm. on compression of both jugular veins. The fluid contained 1 cell and 25 mg. of total protein. Encephalographic examination was performed on August 30. Only 75 cc. of fluid could be obtained. The encephalograms were normal in all respects (fig. 3). After encephalographic examination subtemporal decompression was performed on the right. The brain bulged only slightly through the cranial opening, and no subdural fluid was found. The intracranial pressure appeared pretty well controlled for about three weeks, and the choking of the

optic disks gradually subsided. The intracranial pressure then rose once more and reached the height of 430 mm. of water. Because of this, subtemporal decompression was performed on the left on November 11. At that time, the disks were no longer elevated, and there was secondary atrophy. The vision had improved to 6/20 in the right eye and 1/60 in the left. After the second decompression, the intracranial pressure was only occasionally increased, as evidenced by the bulging at the areas of decompression, and except for the reduction in visual acuity the patient appears at the time of writing to be cured.

It is significant in this case that although the condition had been present for seven months the total volume of cerebrospinal fluid was definitely less than normal. Surely if the clinical picture is due to hydrocephalus, the volume of fluid in this instance should have been much in excess of normal. Without encephalographic examination one might have been tempted to make a diagnosis of hydrocephalus, because it was easy to obtain as much as 48 cc. of fluid by simple spinal puncture.

CASE 5.—A boy of 7 years was admitted to the hospital on May 1, 1935, because of failing vision of four weeks' duration. His history stated that six months previously he had had otitis media on the right, accompanied by headache. There was no drainage from the ear, but three weeks after the onset an abscess in the mastoid was incised and drained. The patient on admission was afebrile and not obviously ill. Physical examination revealed normal conditions except for bulging of the floor and the posterior wall of the right external auditory canal. Examination of the eyes showed advanced secondary optic atrophy, with some edema. The vision was reduced to counting fingers at 1 foot (30 cm.) with the right eye and at 4 feet (120 cm.) with the left eye. Roentgenograms showed evidence of mastoiditis on the right. The spinal fluid pressure was 200 mm. of water. On compression of the right jugular vein there was no rise at all. On compression of the left jugular vein the pressure rose to 300 mm. of water. The fluid contained 2 cells, and the total protein content was 16 mg. Mastoidectomy was performed on the right on May 6. Some granulation tissue was found in the antrum. The lateral sinus was white and lusterless. A needle was introduced into the sinus, but nothing could be aspirated. No further spinal punctures were performed. Three months later the edema of the optic disks had entirely subsided, but there was advanced secondary atrophy. The visual acuity in each eye was 6/60. When the patient was seen two and one-half years later, the findings were the same.

In this case the Tobey-Ayer test definitely indicated an obstruction of the venous outflow on the side of the otitis, and occlusion of the lateral sinus was found at mastoidectomy. The appearance of the optic disks and the relatively low spinal fluid pressure indicated that the process was subsiding, probably because an adequate collateral circulation had developed.

CASE 6.—A boy of 8 years was seen at St. Vincent's Hospital in Toledo, Ohio, on March 8, 1938. Ten weeks previously, he had had influenza, and both ear drums had ruptured spontaneously. After four weeks the drainage ceased on the left side and slowed down considerably on the right. At this time a temperature of 104 F. and signs of mastoiditis on the right developed. Roentgenograms showed

clouding of the right mastoid. The temperature was reduced to normal for a few days by the use of sulfanilamide, but then daily chills and fever developed, accompanied by vomiting and a stabbing pain referred from a point just above the right mastoid to the right frontal region. Choking of the optic disks was found, and the spinal fluid pressure was 22 mm. of mercury. The fluid contained 3 cells. About six weeks after the onset mastoidectomy was performed on the right by Dr. Albert L. Steinfeld. The sinus and dura appeared to be normal. No pus was found in the mastoid, and the culture was sterile. On the day after the operation, however, there was a profuse discharge of pus from the wound, suggesting the possibility that a perisinal abscess had ruptured. After a few days' respite, a spiking fever again occurred. Thrombosis of the left lateral sinus being suspected, mastoidectomy was performed on the left during the tenth week of the illness. There was no pus in the mastoid, and the sinus appeared normal. Two days later the patient complained for the first time of some double vision. He was alert but slightly irritable. There was choking of the optic disks of 4 diopters, with numerous hemorrhages. There was some sluggishness of the patellar and Achilles tendon reflexes in the left leg, but there were no other neurologic signs. The spinal fluid pressure was 47 mm. of mercury. It rose 2 mm. when the right jugular vein was compressed for ten seconds and 6 mm. on compression of the left jugular vein. The right lateral sinus was then reexposed by Dr. Steinfeld and found to be filled with pus. The sinus was widely incised, and the right jugular vein was ligated. The temperature promptly subsided, and five weeks later the patient was well enough to return to school.

In this case also the Tobey-Ayer test indicated the correct diagnosis, so that the surgeon was encouraged to reinvestigate a sinus which at the first operation appeared to be uninvolved.

Case 7.—In a boy aged 7 years, acute otitis media developed on the left on April 5, 1932. Myringotomy was performed within twenty-four hours, followed by the free escape of pus for several days. The patient then complained of mild headaches, and on the fourteenth day diplopia developed. On admission to the hospital the next day, the temperature was normal, and the lad did not appear ill. There were paresis of the right abducens nerve and choking of the optic disks of 4 diopters in each eye. The patient had vomited on one occasion. There were no clinical signs of mastoiditis. A roentgenogram of the skull showed no abnormalities except for some haziness of the left mastoid. The spinal fluid pressure on the sixteenth day was 210 mm. of water. On compression of the right jugular vein the pressure rose to 320 mm. On compression of the left jugular vein it rose only to 240 mm. On bilateral compression it rose to 350 mm. The fluid contained no cells; the total protein content was 20 mg. On the twenty-first day the spinal fluid pressure with the patient under anesthesia induced by avertin with amylene hydrate was 550 mm. of water. The choking of the optic disks had increased, and the visual acuity was beginning to fail. Encephalographic examination on this date revealed 113 cc. of cerebrospinal fluid. The third ventricle was found to be slightly dilated and the sulci only a trifle more prominent than the average (fig. 4). Encephalographic examination was followed by subtemporal decompression on the right. As the dura was carefully opened and before the arachnoid was incised, a quantity of yellowish subdural fluid escaped. The arachnoid was then incised; a quantity of air escaped from the subarachnoid spaces, and the brain retracted from the inner surface of the dura. The wound was closed without drainage. During

his stay in the hospital the patient was subjected to dehydrative measures and frequent spinal punctures. The choking of the optic disks gradually subsided, and he was discharged from the hospital on the forty-ninth day of his illness with a spinal fluid pressure of 220 mm. of water. However, one week later the pressure had increased to 550 mm. of water, so that the following day an encephalogram was again made. On this occasion only 85 cc. of fluid could be obtained. The encephalograms showed the same condition as before, except that the sulci were less well outlined. Dehydrative measures and frequent spinal punctures were continued, and after four and one-half months the spinal fluid pressure was found to be consistently normal and the optic disks flat.

In this case one might question whether sinus thrombosis actually had occurred. The evidence in favor of it is the presence of the sub-

Fig. 4 (case 7).—Third ventricle slightly dilated and the sulci only a trifle more prominent than the average.

dural effusion, plus the rise of only 30 mm. in the spinal fluid pressure when the affected jugular vein was compressed, as compared with 110 mm. when the opposite jugular vein was compressed.

Case 8.—A man of 40 was admitted to Mount Sinai Hospital on April 8, 1931. Four weeks prior to his entry pain in the right ear had developed. The right ear drum was incised, but no pus escaped. Four days later, however, a serous discharge from the ear occurred, which persisted for a few days. On the seventh day of the illness generalized headaches developed, with a thumping sensation in the left parietal region. On the twenty-second day there was transient diplopia. On the twenty-seventh day a chilly sensation and mild somnolence occurred. The next day the temperature was 101 F. The white blood cells numbered 16,000, and the patient was more definitely lethargic. There was a well marked choking of the optic disks but no other significant neurologic findings. The spinal fluid

pressure was 640 mm. of water. The Tobey-Ayer test was not applied. The fluid contained 15 cells. On the twenty-eighth day the right temporal lobe was explored with a cannula, but no abscess was encountered. As the cannula entered the descending horn of the ventricle, 40 cc. of fluid escaped, and the intracranial tension subsided. Encephalographic examination was performed; 120 cc. of fluid was replaced with 140 cc. of air, after which no more fluid could be obtained. The encephalograms showed the lateral ventricles to be exceedingly small but exactly in the midline. The visible cerebral sulci were reduced in size and number. Mastoidectomy was then performed on the right by Dr. Myron Metzenbaum. A large perisinal abscess was uncovered. When this was evacuated, the wall of the sinus was dark. On incision of it there was a gush of blood, and the sinus was packed. The patient's convalescence was satisfactory, and he has remained well at the time of writing.

In this case, if one is to believe that the increased intracranial pressure was caused by occlusion of the sinus it must be assumed either that the pressure of the perisinal abscess caused an obliteration of the sinus or that thrombosis was present beyond the limits of the operative exposure.

CASE 9.—A girl of 12 years was admitted to the hospital on Feb. 27, 1937, because of pain in the left ear, nausea, vomiting and drowsiness. The history stated that she had been swimming three days before and that twelve hours later pain in the left ear developed. The left ear drum was incised, and a considerable amount of thick pus escaped. Because of deepening stupor the patient was brought to the hospital. On admission, the temperature was 103 F. and the pulse rate 62. The patient was stuporous, with periods of restlessness. There was ptosis of the left eyelid, with dilatation of the pupil and external strabismus. There were weakness of the right angle of the mouth and a Babinski sign on the right. Slight nuchal rigidity was present. The clinical diagnosis at the time was acute abscess of the left temporal lobe. The spinal fluid pressure was 420 mm. of water. The fluid contained 8 cells and 30 mg. of total protein. Treatment with prontosil[14] was started on the third day of the illness. On the fifth day the spinal fluid pressure was 300 mm. of water. The fluid contained 15 cells and 25 mg. of total protein. Pus continued to drain from the left ear, but there were no clinical signs of mastoiditis. On the sixth day the patient was definitely improved. She had complete motor aphasia but responded well to commands. Right homonymous hemianopia was present. The discharge from the ear ceased on the twelfth day at which time a roentgenogram of the mastoids showed merely a suspicion of an early stage of mastoiditis. The temperature subsided to normal on the nineteenth day, but the edema of the optic disks had gradually increased to 2 diopters. The spinal fluid pressure was 210 mm. of water. On compression of the right jugular vein the pressure rose in ten seconds to 350 mm.; on compression of the left jugular vein for ten seconds the pressure rose to 240 mm. It was noted at the time that compression of the right jugular vein produced pain in the head, whereas compression of the left jugular vein did not. The patient's progress from then on was satisfactory. On the twenty-sixth day, all neurologic signs had cleared, and the choking of the optic disks was subsiding. The patient was discharged from the hospital after four weeks and has remained well till the time of writing.

14. The disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid. This is the substance previously known as prontosil soluble and now to be called neoprontosil.

In a consideration of this case it may be argued that the results of the unilateral jugular compression test are not convincing, that sinus thrombosis was not present and that the whole picture might be explained by localized encephalitis spreading by contiguity from the disease process in the temporal bone. However, it appears to me more logical to assume that the clinical picture of increased intracranial pressure was due to sinus thrombosis and the localizing signs to extending cerebral thrombophlebitis. The process in this case subsided without suppuration, perhaps because prontosil was used, although the responsible organism was definitely proved to be a nonhemolytic streptococcus.

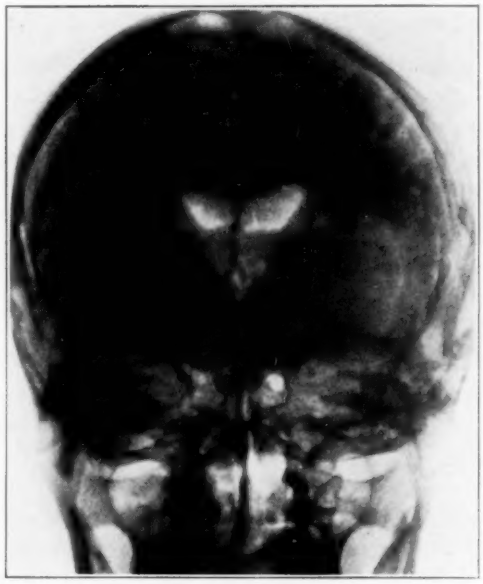


Fig. 5 (case 10).—Normal encephalogram.

CASE 10.—In a man of 25 acute coryza with right-sided headache developed on Feb. 12, 1938. Five days later a discharge from the right ear appeared. Drainage was insufficient; so a myringotomy was performed. Twelve days later mastoidectomy was performed on the right because of diplopia and choking of the optic disks. Pus was found in the mastoid and on the outer surface of the dura. The lateral sinus appeared normal. Convalescence was normal except for continued diplopia and choking of the optic disks.

Eleven weeks from the onset the patient was admitted to the hospital. He had no complaints except blurring of vision and presented every outward evidence of good health. There was 2 to 3 diopters of papilledema in each eye, with hemorrhages on the right disk. Tests for diplopia indicated weakness of both sixth nerves. The visual fields were full except for enlargement of the blindspots. There were no neurologic signs. The spinal fluid pressure was 450 mm. of water. It rose to 520 on compression of the right jugular vein, to 480 on

compression of the left jugular vein and to 560 on bilateral compression. The fluid contained no cells and 20 mg. of total protein. Roentgenograms revealed an operative defect in the right mastoid. The petrous tips appeared normal and were not aerated.

Thirteen weeks from the onset, an encephalographic examination was performed; 175 cc. of fluid was replaced by 185 cc. of air, after which no more fluid could be obtained. Despite the increased amount of fluid, the encephalograms were entirely normal (fig. 5). In order to study the patency of the sinuses colloidal thorium dioxide (thorotrast) was injected into the longitudinal sinus. Because of technical difficulties the roentgen exposure was not accomplished until after all the contrast medium had left the cranial cavity, and the roentgenograms were therefore valueless. Subtemporal decompression was carried out on the left at the time of which not more than the normal amount of subdural fluid appeared to be present. The convalescence till the time of writing has been satisfactory, but sufficient time has not elapsed to permit determination of the end result.

In this case more than any other one might question the diagnosis of sinus thrombosis, especially in view of the negative Tobey-Ayer test. It differs from the previous cases also in that the volume of the cerebrospinal fluid was definitely above normal. In all other respects, however, the disease appears to be the same.

SUMMARY OF CASE REPORTS

Here, then, are presented 10 cases of increased intracranial pressure persisting for from one to eleven months after acute otitis media. In 7 of the cases the patients' ages were between 7 and 12 years. The presence of sinus thrombosis was proved at autopsy or at mastoidectomy in 5 cases and indicated by the Tobey-Ayer test in 3 additional cases. In 1 case a perisinal abscess was found, but the lateral sinus bled when incised. In the remaining case, there was no definite evidence of sinus thrombosis. Encephalograms in 6 cases showed no evidence of an expanding lesion or of hydrocephalus. The total volume of the cerebrospinal fluid was increased in only 1 instance. The cell count in the spinal fluid was elevated in only 2 cases. All patients were treated by repeated spinal punctures with the exception of patient 5, in whom the pressure was only 200 mm. of water and the process was subsiding spontaneously. In 4 cases subtemporal decompression was performed. The intracranial pressure returned to normal in from one to eleven months in 7 cases. In 1 case the pressure is still elevated at the time of writing, three and one-half months after the onset. Necropsy, performed in 2 cases, showed extensive thrombosis of the dural sinuses, including the longitudinal sinus. In 2 cases a subdural effusion was proved to be present. In 7 instances the disease process was on the right side and in 3 on the left side.

In Symonds' experience also this syndrome was found more frequently after otitis media on the right. The explanation of this is found in the anatomy of the dural sinuses. When one of the lateral sinuses receives most or all the blood from the superior longitudinal sinus,

it is more likely to be the right than the left. Therefore, a thrombus of the right lateral sinus is more likely to cause serious impairment of the return of venous blood from the cranial cavity and is also more likely to extend into the longitudinal sinus than is a thrombus in the left lateral sinus.

Additional evidence that interference with the return of venous blood from the cranial cavity can cause increased intracranial pressure is the occasional occurrence of choked disks after ligation of the jugular veins or after accidental laceration and packing of the lateral sinus during mastoidectomy. Williams [7] (case 2) and Davidoff and Dyke [5] (case 9) each described such an occurrence.

COMMENT

I should like to call attention to some false interpretations of certain observations which have been made in cases of this type. First of all, the large amount of cerebrospinal fluid obtained at lumbar puncture before the pressure is reduced to normal does not indicate an increase in the amount of cerebrospinal fluid present. Because the venous outflow is obstructed, the spinal fluid removed is immediately replaced by an increase of venous blood within the already engorged cerebral veins. This engorgement of the cerebral veins forces a larger quantity of cerebrospinal fluid out of the lumbar puncture needle than one would be able to obtain if the venous obstruction were not present. Every one knows that a larger quantity of fluid can be obtained at spinal puncture if one applies jugular compression to increase the pressure and the rate of flow. A similar factor is operative in these cases.

Another frequent observation in this type of case is that when trephination is performed the exposed portion of the arachnoid membrane is found to be distended with fluid. This observation has led the surgeon to assume that external hydrocephalus is present. This, however, is not the case, at least in the majority of instances. Since encephalograms in these cases are essentially normal, it seems fair to assume that the subarachnoid space is distended as disclosed at trephination because the release of pressure at the site of operation produces local accumulation of fluid in the subarachnoid space at this area. In other words, in these cases the cerebrospinal fluid is normal in amount and circulates freely, so that when the increased pressure is released at a localized area, as in decompression, it rapidly follows the subarachnoid channels to that point, causing local distention of the subarachnoid spaces.

Still a third observation which has led to the belief that hydrocephalus is present in these cases is the copious flow of fluid that occasionally occurs when a cannula is introduced into the lateral ventricle. This has been interpreted as meaning that the ventricles are dilated. That this assumption is incorrect is shown by encephalographic examina-

tion, which demonstrates that in these cases the ventricles are normal in size or even smaller than normal. When one can obtain a large quantity of fluid from the ventricle in a case of this sort it simply means that, since the cerebrospinal fluid system is not obstructed, the expanding intracranial veins squeeze all the fluid toward the point of exit. Therefore, toward the end of the drainage the fluid that is escaping from the cannula is really subarachnoid fluid, which has been forced into the ventricle as a result of the drainage. In these cases one cannot estimate the size of the ventricles from the amount of fluid obtained by ventricular puncture.

In conclusion, therefore, I am in entire agreement with Symonds that the syndrome of increased intracranial pressure without localizing signs occurring after an attack of otitis media is usually due to sinus thrombosis, and that when this picture of increased intracranial pressure is associated with localizing cerebral signs and recovery occurs without suppuration it means that the thrombus (perhaps sterile) within the intracranial sinuses has extended into the cerebral veins. I am not convinced, however, that the thrombus must necessarily extend into the sagittal sinus in order to produce increased intracranial pressure. If, as is not infrequently the case, one of the lateral sinuses constitutes the chief emissary for the intracranial circulation, then I believe it is possible for a thrombosis of this sinus alone to produce the picture of increased intracranial pressure. Since the total volume of the intracranial fluid is usually not increased and since encephalograms in these cases are essentially normal, there obviously is no hydrocephalus. I am inclined to attribute the increased intracranial pressure simply to engorgement of the intracranial veins, plus, in some cases, sterile subdural effusion resulting from external pachymeningitis.[15] Recovery from this state of increased intracranial pressure occurs when, by means of recanalization or the development of collateral supply, the return of venous blood from the cranial cavity is once more adequate. The chief value of subtemporal decompression in these cases is not that it causes a satisfactory reduction of the intracranial pressure but that the degree of tension in the hernia serves as a guide as to when a therapeutic lumbar puncture should be done. Another important reason for decompression is that it permits the evacuation of a subdural effusion if one is present. Instead of referring to this syndrome by a variety of names it appears preferable to adopt a descriptive title, such as "sinus thrombosis causing intracranial hypertension."

15. T. H. B. Bedford (Effect of Increased Intracranial Pressure on the Pressure of the Cerebrospinal Fluid, Brain **58**:427-447 [Dec.] 1935) has demonstrated by accurate experiments on animals that, although the torcular venous pressure remains elevated after ligation of the chief venous emissaries, the cisternal pressure returns to normal in about an hour. The results of these acute experiments on the laboratory animal, however, do not by any means prove that obstruction of the major intracranial sinuses in man may not result in persisting increase in intracranial pressure.

ACUTE INFECTION OF THE CIRCUMVALLATE PAPILLA

RICHARD WALDAPFEL, M.D.
GRAND JUNCTION, COLO.

Little is known about acute inflammations of the papillae of the tongue. From a clinical, anatomic and pathologic standpoint these infections are usually included with diseases of the mucous membrane of the tongue and are not separated from the various types of glossitis.

The fact that such isolated infections occur and that they offer diagnostic difficulties will be discussed in this article. The various papillae of the tongue (fig. 1) offer a variety of conditions for the harboring of infectious organisms, according to their anatomic structure. The filiform papillae are the smallest and most numerous ones; they are spread above the dorsum of the anterior portion of the tongue and give the tongue its velvet-like appearance. Histologically, they are smooth-walled continuations of connective tissue which are lined with stratified squamous epithelium. The fusiform papillae are single, larger mushroom-shaped, club-shaped or wartlike elevations among the filiform papillae and have the same histologic structure. There are no cavities and no possibilities of retention in either type of papilla. The foliate papillae are two single papillae situated on the posterior margin of the tongue on either side. They are elevations consisting of several folds of mucous membrane which are separated by deep fissures. A retention of infectious organisms might occur in the deeper recesses of these papillae and a pathologic process develop, especially after trauma or injury due to decayed teeth. Such decubital infections are clinically known, and the disease is one of the few of the papillae which are described in the textbooks.

The vallate papillae (fig. 1) consist of seven to twelve large elevations which are arranged in a V-shaped line, separating the anterior two thirds of the tongue from the posterior one third. The open angle of this line looks forward, and the point of the V lies in front of the foramen caecum, which is the uppermost part of the former thyroglossal duct. Each of these papillae may be regarded as a conus, the apex of which is sunken rather deeply into the tongue; so far, in fact, that the base is but slightly higher than the surrounding surface of the tongue. Each papilla (fig. 2) is surrounded by a deep circular fossa, which, like the

Presented before the Colorado Oto-Laryngological Society, Denver, Feb. 4, 1939.

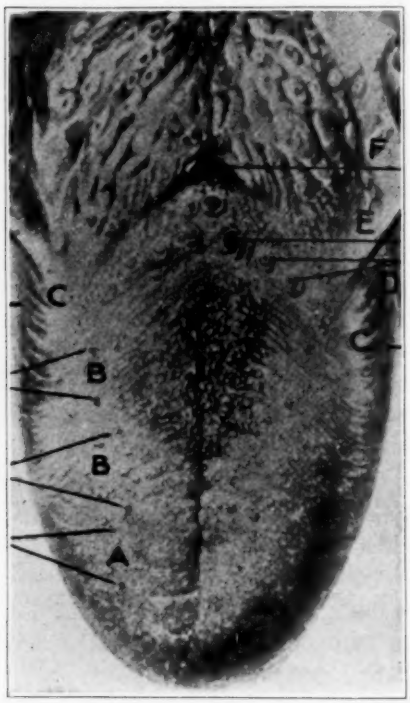


Fig. 1.—The normal tongue. Filiform papillae are indicated by *A*; fungiform papillae, by *B;* foliate papillae, by *C;* circumvallate papillae, by *D;* the first circumvallate papillae left of the midline (the one affected in the reported case), by *E,* and the foramen caecum by *F.*

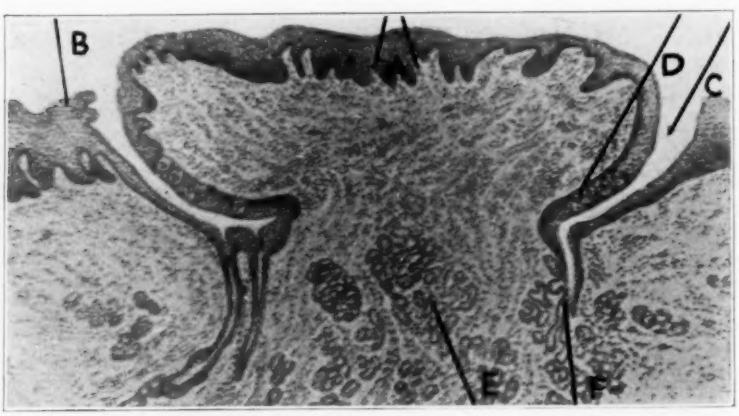


Fig. 2.—Microscopic section through a circumvallate papilla. Secondary papillae are indicated by *A*; squamous epithelium, by *B*; the circular fossa of the papilla, by *C;* taste buds, by *D;* serous glands, by *E,* and the excretory duct of the serous gland by *F.*

papilla itself, is lined with stratified squamous epithelium containing hundreds of taste buds. The body of the papilla is formed from connective tissue, and sometimes two or three papillae are surrounded by a common fossa.

The factors which differentiate these circumvallate papillae from the others are (*a*) the deep circular fossa and (*b*) the opening of numerous serous glands into this fossa—an important anatomic feature. These two features, both anatomic, obtain their adult structure from the embryologic structure (fig. 3). These papillae are formed from a downgrowth of epithelium into the mesoderm in the shape of a ring. The downgrowth becomes hollowed out, and there is an additional outshoot or branch from it; the mucous glands originate in the latter branch, which also becomes hollow and serves as the duct for the mucous glands; all of these glands communicate with the ring (fig. 3). The entire papilla thus develops as a unit together with the gland. These two anatomic features unquestionably are responsible for the introduction of infection

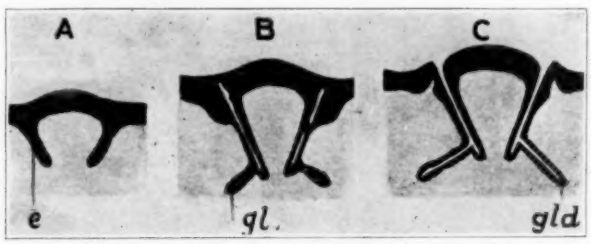


Fig. 3.—Development of a circumvallate papilla (after Gråberg, J.: Beiträge zur Genese des Geschmacksorgans des Menschen [figs. 2, 3 and 4], Morphol. Arb. **8**:117, 1898). In *A* the epithelial downgrowth (*e*) is shown projecting into the mesoderm in the form of a ring. In *B*, the downgrowth becomes hollowed out; a branch projects farther into the mesoderm, forcing the anlage of the mucous glands (*gl.*). In *C*, these become hollowed out, forming the ducts (*gl. d.*) of the mucous glands and communicating with the circular fossa of the papilla.

in these papillae. A comparison of such an infection might be made with an infection of the skin wherein the hair follicle and contiguous sebaceous gland become infected; the sebaceous gland is a part of the schematic arrangement and a furuncle results.

REPORT OF A CASE

An infection of the vallate papilla therefore leads to the clinical picture described in the following case report.

Mrs. J. S., aged 50, reported to the office on Dec. 12, 1938. She stated that for the past three days she had had difficulty in swallowing and that the condition was constantly becoming worse. She also stated that she had had a similar infection on two previous occasions, two months and six months prior to presenting

herself at the office. On these two previous occasions the condition was not so severe. Her chief complaint at the time of examination was "terrible pain in the throat."

Examination gave the following information: The temperature was 99.2 F., the respiratory rate 96 and the pulse rate 22. The cervical glands were not swollen; the sublingual area was normal, and the tongue showed a grayish yellow coating and was not particularly visibly swollen. The tonsils had been cleanly enucleated, and parts of the left anterior and posterior pillars were covered with whitish membranes; which were adherent to the mucous membrane. The epiglottis was free and movable. The slightest pressure on the tongue elicited pain, and when the patient was asked to swallow the pain was markedly manifest. Other than the coating of the tongue and the membranous areas, no pathologic changes were observed, and the possibility of pemphigus was considered.

Because of the extreme pain and inability to swallow, the patient was given suppositories containing opium, which partially alleviated the pain, fluids by rectum, an oral antiseptic and short wave diathermy through the upper cervical region.

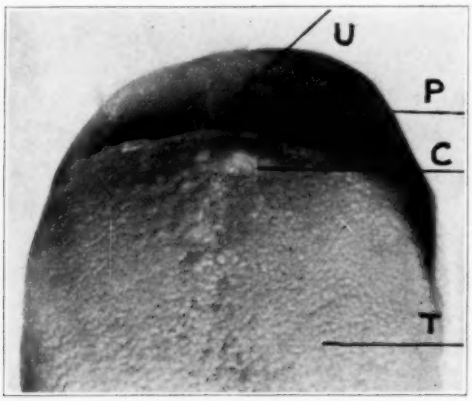


Fig. 4.—Clinical picture of the infection of the circumvallate papilla. The tongue is indicated by *T*; the infected circumvallate papilla, by *C*; the anterior pillar, by *P*, and the uvula and soft palate, by *U*.

On December 13 she stated that during the night she had expectorated a quantity of pus with a bad fetor and had immediately felt relieved. On examination the tongue was found to be partially free from the coating, and a red circumscribed swelling the size of a split pea was distinctly seen at the beginning of the posterior third (fig. 4). Thorough examination showed that the swelling corresponded to the first circumvallate papilla left of the midline (fig. 1 *E*); all of the other circumvallate papillae were pale and normal in size. The involved papilla was reddened, enlarged and tender, and on pressure on the middle third of the tongue pus and serous secretion oozed from the center of it. A probe introduced into the papilla revealed a groove a few millimeters in depth. The membranous areas on the pillars and posteriorly were much reduced in size and less tenacious. On December 14 the patient felt almost normal; the swelling of the papilla was greatly reduced, there was no secretion on pressure and the membranous areas were cleared up. Three days later the patient reported by phone that she was normal again.

COMMENT

This case represents one of an infection of a circumvallate papilla, with retention and abscess formation. This superficial process alone could not explain the serious condition and the severe symptoms of the patient. One must consider that the infection must lie deeper in the anlage, similar to the cutaneous infections previously mentioned in which the sebaceous glands were involved. In other words, this infection must have progressed in a retrograde manner from the circular fossa into the opening of the serous glands. In cases of a serious nature there exists at the same time a severe inflammation of one or more of the serous glands of the circumvallate papillae. The quantity of pus which was expectorated and expressed by the patient subsequently verified this opinion; the symptoms could not have been explained by an infection of the small cavity of the papilla.

Every severe process of this kind should be regarded as an isolated infection of a circumvallate papilla and its own related identical serous gland, which appears as a unit embryologically, anatomically and pathologically. The mode of infection is not known; perhaps injuries through foreign bodies in the food may play a part.

The diagnostic difficulties arise mainly in the beginning of the disease and are due to the thick coating on the tongue and adjacent mucous membrane areas which covers the portal of entry of the infection and the diseased area. This coating develops especially easily when the patient is not eating normally and clears up automatically afterward.

Points which aid in making the differential diagnosis are:

1. Extreme tenderness of the tongue is manifest at the slightest touch without evident reason. (This area is especially rich in nerve endings, there being three areas of nerves, the sensoric lingual, the glossopharyngeal and the vagus nerves.) The tongue is movable and apparently not greatly swollen, and the floor of the mouth is free from infection.

2. The base of the tongue is free from involvement, and there is no laryngeal edema present, because the whole tough base of the tongue lies between the larynx and the affected part of the tongue.

3. The temperature is only moderately high, which is different from that associated with other abscesses of the tongue or phlegmons of the floor of the mouth, and is not in keeping with the severe clinical picture of the disease.

In making a diagnosis, eventually the possibility of an abscess formation in a mucous cyst or in a cyst of the thyroglossal duct comes up; both can be ruled out by history and by the local findings.

The prognosis is good. Heat (ultrashort wave diathermy) and antiphlogistic applications break down the papilla and spontaneous discharge results; if the condition is of long duration and fluctuation of the tongue is discernible, a superficial incision is advisable.

SUMMARY

A well circumscribed and rather unknown clinical entity exists which must be regarded as an abscess of a circumvallate papilla and its own identical gland. The knowledge of the condition is important and justifies the anatomic and clinicopathologic description given here.

Case Reports

BACILLUS PROTEUS IN OTOGENIC INFECTIONS, SECONDARY MASTOIDITIS, THROMBOSIS OF THE LATERAL SINUS AND BACTEREMIA

BENNETT G. GERZOG, M.D., BROOKLYN

The infrequency of the occurrence of Bacillus proteus in otogenic infections and the high mortality which it causes make it of sufficient interest to justify reporting the present case.

Since 1912 only 9 cases have been reported in the literature. Seven of the patients gave a history of chronic otitis; all were operated on and 4 had the lateral sinus obliterated and the internal jugular vein ligated. The blood cultures were positive in all but 1 case, and that was the only case in which the patient recovered. The mortality was 89 per cent; 3 patients died of meningitis and 5 of the overwhelming bacteremia.

REVIEW OF THE LITERATURE

Ross [1] in 1912 reported the case of a boy aged 9 months who died after an illness of about six days. There had been typical signs and symptoms of meningitis. From the spinal fluid organisms were isolated. Both ears contained pus. Necropsy revealed a thick layer of greenish yellow pus covering the entire surface of the brain and the spinal cord. The ventricles were dilated with purulent fluid.

Bauer [2] in 1918 mentioned a fatal case of meningitis secondary to otitis media. He gave no detailed description of the history but a detailed account of the morphology and pathogenicity of B. proteus.

In 1922 Kernan [3] described 2 fatal cases of infection with B. proteus. The first patient had a peritonsillar abscess, which was followed by thrombosis of the internal jugular vein, the petrosal and the cavernous sinus and the ophthalmic veins.

The second patient was a man who was admitted to the hospital with a history of sudden onset of headache and indigestion. His symptoms were those of severe sepsis—chills, fever, sweating and at times delirium. He lost 30 pounds (13.6 Kg.). He had had a discharging right ear for seven years. There was no exacerbation of the aural symptoms at the beginning of the illness. On admission the drum of the right ear was seen to be totally destroyed and replaced by granulation tissue. A blood culture contained B. proteus. The next day simple mastoidectomy was performed. A cholesteatoma was found in the antrum. The sinus plate was necrosed, and over the sinus was found

1. Ross, E. A.: Bacillus Proteus Meningitis, Lancet **2**:1292, 1912.

2. Bauer, A.: Méningite purulente aiguë causée par un proteus (Bacillus submobilis), Compt. rend. Soc. de biol. **81**:745, 1918.

3. Kernan, J. T.: Two Cases of Infection by Bacillus Proteus, Laryngoscope **32**:304, 1922.

a large epidural abscess which extended well down to the bulb and back toward the torcular herophili. This abscess consisted of foul greenish pus. The lateral sinus was obliterated and the internal jugular vein ligated. The patient received numerous transfusions, but the course became increasingly unfavorable, and death occurred ten days after the operation.

Kortenhaus [4] in 1930 operated on a man aged 56 who had had a discharge from the ear for thirteen years and an acute condition for fifteen days, with pain in the ear, foul-smelling discharge, headache, chills and fever. The patient had been admitted to the hospital in a moribund condition, and he died on the night of the operation. The operative findings were a large cholesteatoma in the antral region, perisinal abscess with foul-smelling pus and sinus phlebitis. The diagnosis on admission was otitic meningitis.

The only case of recovery was that described by Simon Stein [5] in 1930. A boy 18 years of age was referred to him because of swelling and pain in the region of an old mastoidectomy wound. He had had severe rhinitis for a few days, with pain in the right ear and an increase of the chronic discharge which had followed the previous mastoidectomy. After several sleepless nights swelling and redness appeared over the old scar. On incision of the scar there was a gush of foul-smelling pus. The lateral sinus was completely exposed except for dirty gray patches of granulation tissue. Culture of the pus showed B. proteus. Wet dressings with diluted solution of sodium hypochlorite were applied for a few days, and this treatment was followed by daily application of dressings with a 1 to 5,000 solution of permanganate. After seven weeks of illness the patient made an uneventful recovery.

Dr. Stein's patient did not present the picture that usually accompanies infection with B. proteus. His condition was not toxic; the blood culture was sterile, and septic fever was lacking.

In 1935 Pavey-Smith and Miller [6] reported the death of a young nurse who came under their care. The patient was admitted to the hospital with a temperature of 104 F.; she gave a history of the extraction of a tooth two days prior to admission. The tooth socket appeared to be clean, and there was no swelling of the alveolus or the face. The temperature remained high, and four days later a slight facial weakness appeared on the left side. The patient made no complaint of pain or discharge from the left ear and stated that she was not deaf but had had aural trouble as a child. The right ear was normal; on the left side there were marked middle ear deafness, granulations in the region of the attic and a small amount of pus in the fundus, but no swelling or tenderness over the mastoid. A mastoidectomy was performed, and foul-smelling pus was found. A large area of the lateral sinus lay

4. Kortenhaus, F.: Proteusmeningitis vom Ohr her, Ztschr. f. Laryng., Rhin. **19**:146, 1930.

5. Stein, S.: Bacillus Proteus Vulgaris Infection in Secondary Mastoiditis, Following Tonsillectomy, and in Radium Implantation in Tonsils, Ann. Otol., Rhin. & Laryng. **39**:1155, 1930.

6. Pavey-Smith, A. B., and Miller, S.: Lateral Sinus Thrombosis Due to Bacillus Proteus, Lancet **1**:862, 1935.

exposed and discolored. Exploration of the lateral sinus showed foul-smelling pus of similar character; the sinus was obliterated and the internal jugular vein ligated. The temperature remained high, and neither repeated blood transfusions nor administration of antistreptococcus serum intravenously appeared to influence the course of the disease. Three weeks after admission the patient died.

In 1936, from the University of Virginia Hospital, McGovern[7] reported 2 fatalities from otogenic infection with B. proteus. Both patients were children, aged 9 and 13 years, and both presented a history of chronic otitis. The operative findings were almost identical: foul-smelling pus, perisinal abscess and sinus phlebitis. The first patient died of the severe septicemia, while the second died of pulmonary hemorrhage after the development of pneumonia due to infection with B. proteus.

The most recent case was reported by Vallee and Giroux.[8] A man aged 25, admitted to the hospital with a history of chronic otorrhea, for the past fifteen days had been complaining of pain in the right ear, right hemicrania, diarrhea and septic fever. Simple mastoidectomy was performed, and a large granuloma in the antral region was found. The sinus was also found to be inflamed and covered with foul-smelling pus. A few days later the patient died. Autopsy showed the lungs to be studded with multiple abscesses, with empyema on the right. Fifteen hundred cubic centimeters of foul-smelling pus was evacuated.

Blood cultures in all the reported cases with the exception of Dr. Stein's were positive for B. proteus.

REPORT OF A CASE

M. W., a girl aged 12, was admitted to the Beth-El Hospital on June 11, 1938, with the complaint of pain in and behind the left ear, frontal headache, left hemicrania and intermittent chills and fever (105 F.) for the past four days.

History.—The family history was noncontributory. Delivery and infancy had been normal. The patient had had pertussis at 6 years of age, measles at 7 years and simple mastoidectomy on the left at the age of 8. The left middle ear remained unhealed.

Four days prior to admission, the child began to complain of pain in the left ear and in the retroauricular region, which radiated to the temporoparietal area. The temperature on that day rose to 105 F., and the patient complained of a chilly feeling. The next day the symptoms somewhat receded, but they increased again in the following twenty-four hours, during which the condition was characterized by severe chills, high temperature and nonprojectile vomiting. There was no aural discharge.

Physical Examination.—A white adolescent girl, well developed and well nourished, showed dusky cyanosis of the face and lips and appeared acutely ill. She complained of severe earache and headache. The head showed no tenderness except over the mastoidectomy scar. There was no pus in the left auditory canal.

---

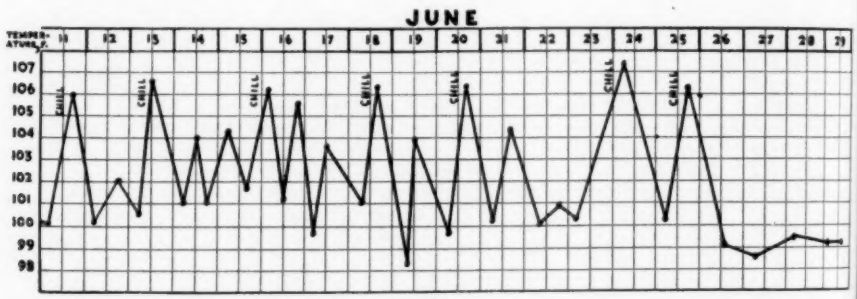
7. McGovern, F. H.: Fatal Otogenic Infection with Bacillus Proteus: Report of Two Cases, Arch. Otolaryng. **24**:618 (Nov.) 1936.

8. Vallée, A., and Giroux, M.: Mastoidite aiguë à Proteus vulgaris: Septicémie, pleurésie, Laval méd. **2**:107, 1937.

The tympanum was partly destroyed and the middle ear filled with granulations. The right ear was normal. Ocular examination showed the veins of the left fundus to be slightly engorged and the margins of the disk to be indistinct. The tongue was dry, inflamed, edematous and heavily coated. The nasopharyngeal mucosa was deeply injected. There was slight nuchal rigidity, with anterior cervical adenopathy on the left. The lungs were normal and the heart sounds good. The abdomen and extremities were normal.

A diagnosis of secondary recurrent mastoiditis and thrombosis of the lateral sinus was made and immediate surgical intervention recommended.

Operation.—A postauricular incision through the previous scar was made, and a completely regenerated cortex was exposed. The mastoid was repneumatized and the intercellular spaces filled with green, foul-smelling pus. The lateral sinus was exposed, inflamed, discolored and covered with granulations. It was decided to obliterate the sinus, and on incision of its wall the lumen was found to be filled with the same foul-smelling pus. Drains were inserted in both the end near the torcular Herophili and the bulbar end and the mastoid cavity packed with iodoform gauze. The internal jugular vein was also ligated. Specimens of pus from the



Temperature chart showing the patient's reaction to the bacteriophage.

mastoid and the lateral sinus were sent to the laboratory. A blood culture was also taken.

Clinical Course.—The following day the temperature dropped to 100 F. and it remained low for twenty-four hours. However, the child appeared restless and complained of considerable pain. Supportive measures were instituted, and a continuous clysis of 5 per cent dextrose in a saline solution was given. Another blood culture was taken.

On June 14 the laboratory reported that the pus from the mastoid and the blood culture yielded a pure growth of B. proteus.

On the basis of the bacteriologic findings the patient was immediately given treatment with a stock bacteriophage which was obtained from Dr. W. J. McNeal's laboratory at the New York Post-Graduate Medical School and Hospital. The bacteriophage was given intravenously for the next seven days, 1 cc. in 1:10 dilution being given at forty-five minute intervals until 10 cc. had been given and then 1 cc. of the undiluted serum every forty-five minutes until 40 cc. had been administered. The dose varied from day to day with the patient's reaction, which was characterized by severe chills and extremely high temperatures, sometimes as high as 107 F. Blood cultures were taken at forty-eight hour intervals in the interim.

The first signs of improvement were noticed on June 21, ten days after operation. The child's condition appeared less toxic, and she began to notice her environment. The tongue was moist and clean and the pulse regular and of good quality. Large quantities of fluids were requested. The blood culture made on June 17 was reported negative. The lateral sinus still drained pus, and because of that the administration of bacteriophage was continued for another four days.

On June 25 the temperature, which had been continuously high, suddenly dropped to normal, and it remained below 100 F. until the day of discharge, July 9. Because of extreme asthenia two transfusions of 200 cc. of citrated blood were given, one on June 25 and the other three days later.

The lateral sinus ceased draining pus about July 5, and from that time on granulations were allowed to form in the mastoidectomy cavity.

Laboratory Findings.—Cultures of pus from the mastoid and the sinus and of blood contained B. proteus. Blood counts were as follows:

Date	Erythrocyte Count	Hemoglobin, %	Leukocyte Count	Polymorphonuclears, %	Mononuclears, %
June 11	3,700,000	75	8,600	86	14
June 15	3,800,000	68	15,950	80	20
June 30	3,600,000	75	9,000	78	22

Subsequent cultures of pus from the mastoid and the lateral sinus contained B. proteus. Blood cultures made on June 11, 13 and 16 contained B. proteus; those made on June 17, 18, 20 and 25 were negative. On June 11 the spinal fluid was under normal pressure, appeared clear, contained 40 cells and yielded a sterile culture. On June 14 it was normal. Examination of the urine at frequent intervals revealed nothing of significance. Cultures for B. proteus were always negative.

COMMENT

Otogenic infections with B. proteus are infrequent and usually fatal.

They are usually found, if at all, in chronically infected ears and therefore may be considered a secondary rather than a primary invader.

The organism causes slow, progressive, putrefactive necrosis with extreme toxemia.

The organism seems to have an affinity for vascular walls, since 50 per cent of the patients have had necrosed sinuses and 1 patient died of pulmonary hemorrhage.

Ross [1] cited Sewall's statement that autogenous vaccines have been suggested and found to be effective on several occasions. I have failed to find further details concerning that form of therapy.

It is probable that the successful outcome in the case here reported may be attributed to the use of the bacteriophage, and it is suggested that this be used freely in other cases of similar infection.

SEQUESTRATION OF THE STYLOID PROCESS COMPLICATING A PARAPHARYNGEAL ABSCESS

HERMAN RUBIN, M.D., AND SAMUEL GALBURT, M.D., BROOKLYN

A review of the literature does not reveal the record of a case similar to that which is being reported.

REPORT OF A CASE

A white woman aged 38 was admitted to the hospital on Oct. 29, 1935. She presented the following history: A week before admission two molar teeth were extracted from her left lower jaw. On the following day, a swelling appeared on the right side of her neck, extending from the chin to the angle of the jaw.

Six days later, at the time of admission, she presented a swelling in the floor of the mouth, on the right side, which was pushing the tongue upward. There were tenderness and swelling in the neck on this side. The temperature ranged between

Sequestration of the styloid process.

102 and 104 F. for several days. The blood count was as follows: red cells, 4,210,000; hemoglobin content, 83 per cent; white cells, 25,000; neutrophilic polymorphonuclears, 88 per cent; lymphocytes, 5 per cent; monocytes, 7 per cent. The urine revealed traces of albumin and sugar. The blood culture was negative, as was the Wassermann reaction of the blood.

Operation.—With the patient under local anesthesia, an incision 2 inches (5.1 cm.) long was made parallel to the body of the mandible. An abscess in the submaxillary space containing an ounce (29.6 cc.) of foul-smelling pus was evacuated. The presence of hemolytic streptococci was revealed by the culture.

Postoperative Course.—The swelling in the neck subsided. The tongue receded, and the temperature returned almost to normal. On the fifth day after operation, the temperature rose to 103 F. The patient complained of soreness in the throat and pain along the right side of the neck. Examination revealed the following condition: The right tonsil protruded; some trismus was present, and the parotid gland was slightly tender and more prominent than is normal. Tenderness and swelling were noted along the anterior border of the sternocleidomastoid muscle.

We now felt that we were dealing with an infection of the submaxillary space extending into the parapharyngeal region. The previous incision was carried

From the otolaryngologic service of Dr. Louis C. Menger, Bushwick Hospital.

downward along the anterior border of the sternocleidomastoid muscle, and about ½ ounce (15 cc.) of foul-smelling pus was evacuated from the parapharyngeal space. The temperature returned to normal, and the patient was discharged five days later.

She returned two weeks after she was discharged, with a temperature of 101 F., complaining of pain on swallowing. This had lasted several days, and she now had difficulty in opening her mouth. The right tonsil was pushed forward, and a marked bulge was noticed in the peritonsillar region. This was incised, and 2 drachms (7.4 cc.) of greenish yellow pus was obtained.

The peritonsillar region continued to discharge a small amount of pus. It was therefore thought advisable to perform a tonsillectomy. This was done with the patient under local anesthesia two months after her initial symptoms began. On removal of the tonsil, a cone-shaped sequestrum of bone was found lying in the retrotonsillar space. This sequestrum was 2 mm. in diameter and 14 mm. long. It was identified as the right styloid process. The patient made an uneventful recovery, except for slight stiffness of the jaw.

Comment.—The patient came to us with cellulitis of the floor of the mouth on the side opposite to that from which the molar teeth had been extracted. This was probably due to an abrasion from dental instrumentation. The infection extended to the parapharyngeal space and probably caused osteomyelitis of the styloid process, which resulted in its sequestration. The styloid process divides the parapharyngeal space into an anterior and a posterior compartment. It can readily be seen how a sequestrum of this structure could make its way out of this space to cause an abscess and swelling of the peritonsillar or the retrotonsillar region. Removal of the tonsil in this case disclosed the sequestrum and cured the peritonsillar suppuration.

277 Eastern Parkway.

1529 West Sixth Street.

Clinical Notes

A PRACTICAL METHOD FOR POSTERIOR POLYPECTOMY

E. R. Hargett, M.D., Springfield, Ohio

The mechanical difficulties of polypectomy in the anterior parts of the nose are usually well handled by the snare method. However, the wire is not so easily manipulated in the middle and posterior portions, and it is to improve the operative technic in these parts of the nose that the following suggestions are given.

A, Noyes's alligator action, serrate-jawed forceps. *B*, Williams' bayonet perforator.

The polyp is cleaned and defined and possibly maneuvered into a better position by the liberal use of point suction. A eustachian catheter attached to a suction tube serves well for this purpose. The polyp is grasped near its base with a Noyes alligator 7 inch (17.8 cm.) forceps, as much of the base as possible being included. At this point some operators remove the growth or the portion of it grasped by a twisting, tearing maneuver that may injure the mucous membrane of the nose or even the underlying ethmoid sinuses. By contrast, my technic involves cutting through the base with a Williams bayonet perforator. It will be found that the construction of this ear knife lends itself nicely to visual manipulation along the edge of the Noyes forceps, an assistant holding the nasal speculum. The grasping and incising can be repeated after more point suction if the growth has a wide base.

The method is less painful than other methods, there is less bleeding, healing is quicker and the patient does not get the impression of crudeness.

It is well to keep the blade of the knife toward the midline.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

---

TUMORS OF THE NOSE AND THROAT

GORDON B. NEW, M.D.
ROCHESTER, MINN.

WALTER KIRCH, M.D.
DES MOINES, IOWA
AND
JOHN B. ERICH, M.D.
ROCHESTER, MINN.

Welch and Nathanson [1] presented some interesting observations on the incidence of malignant diseases of the lip, oral cavity, antrum, pharynx and larynx and on the expectancy of life of persons suffering from them. The data for this paper were obtained from the Huntington and Pondville hospitals of Wrentham, Mass., and include all patients admitted before the year 1933. In addition to data on sex and age incidence, the authors introduced statistics concerning the median expectancy of life of untreated and treated patients who had malignant diseases of the before-named regions of the body. The expectancy of life for untreated patients with intraoral carcinoma was ten months; for those with carcinoma of the larynx, twelve months. For treated patients with malignant lesions of the lip the median expectancy of life was sixty-six months; of the buccal mucosa, twenty-four months; of the palate, eighteen months; of the upper alveolus, twenty months; of the lower alveolus, eighteen months; of the tongue, seventeen months; of the floor of the mouth, seventeen months; of the tonsil, fifteen months; of the pharynx, fourteen months; of the antrum, seventeen months, and of the larynx, eighteen months. The median expectancy of life with all intraoral carcinomas was seventeen months for men and twenty-one months for women.

From the Section on Laryngology, Oral and Plastic Surgery, the Mayo Clinic.

1. Welch, C. E., and Nathanson, I. T.: Life Expectancy and Incidence of Malignant Disease: Carcinoma of the Lip, Oral Cavity, Larynx and Antrum, Am. J. Cancer **31**:238-252 (Oct.) 1937.

In some general observations on the treatment of malignant disease of the upper respiratory and digestive tracts, Young [2] declared his preference for radium or roentgen therapy rather than surgical treatment. However, he stated that for malignant growths of the intrinsic larynx surgical treatment is the method of choice and that fenestration is a better surgical procedure than is laryngofissure. His tables concerning the results of treatment are interesting but not entirely conclusive, since the time after operation or irradiation was not indicated.

Charteris [3] described the treatment of malignant neoplasms of the mouth, nose and throat by radium applied locally over the lesion or inserted into it. For carcinoma of the tongue, he stated, the implantation of radium yields excellent results as far as the local lesion is concerned. However, he expressed the opinion that cervical metastases are particularly difficult to treat and concluded that block dissection of the cervical glands is the most effective method of therapy. Faucial malignant neoplasms respond well to radium, he indicated; his tables demonstrate that 10 per cent of his patients treated in 1930 survived five years and 25 per cent of those treated in 1931 survived four years. For hypopharyngeal, postcricoid and extrinsic carcinomas, he concluded that the local use of radium is ineffective and that the employment of external irradiation is preferable. Charteris' tables demonstrated good results in the management of intrinsic carcinomas of the larynx by means of fenestration and the insertion of radium. His treatment of esophageal malignant growths with a Souttar tube, which has a central gold tube containing radon and which is suspended in the esophagus, was successful in a few cases only. For carcinomas of the nasal sinuses, Charteris' best results were obtained by opening the sinus, surgically removing all the visible tumor and finally inserting radium.

Kriegsmann [4] reported 62 cases in which malignant tumors of the upper respiratory passages were treated by the Coutard method of irradiation. Of the 62 tumors, 20 were situated in the epipharynx (including the nose and paranasal sinuses), 14 in the mesopharynx and 12 (including extrinsic growths of the larynx and tumors of the esophageal introitus) in the hypopharynx; 16 were intrinsic in the larynx. With tumors of the first group, the only patients that survived without recurrence from a year and a half to seven years were 1 with a carcinoma of the turbinate and 1 with a similar lesion of the ethmoid sinus; both

2. Young, G.: Some Remarks on Malignant Disease, J. Laryng. & Otol. **52**: 477-483 (July) 1937.

3. Charteris, A. A.: The Use of Radium in Certain Malignant Conditions of the Throat and Nose, J. Laryng. & Otol. **52**:484-491 (July) 1937.

4. Kriegsmann: Zur Röntgenlangzeitbehandlung maligner Tumoren im Bereich der oberen Luftwege, Ztschr. f. Hals-, Nasen- u. Ohrenh. **42**:385-396, 1937.

were treated by surgical diathermy together with irradiation. Treatment of the second group, comprising exclusively carcinomas of the tonsil, resulted in three to eight year cures in 5 cases. Combined diathermy and irradiation was again found to be the most efficient. Only 1 patient with a hypopharyngeal tumor remained free of symptoms at the end of two years. This group was considered to be particularly unfavorable for treatment. The best results were obtained for patients with intrinsic laryngeal tumors, of whom 8 survived symptom free from two to seven years. Kriegsmann concluded that Coutard's method offers as much as surgical treatment but that when conservative surgical treatment is applicable it is less exhausting to the patient.

NOSE

Mesial dermoid cysts of the nose occurring in twin boys were reported by Furniss.[5] The development of such cysts, he concluded, is due to a failure of coalescence of the two median nasal processes, which normally become united during the second week of fetal life.

In discussing epitheliomas of the nasolabial fold, Meland[6] stated that such growths extend along the various embryonic lines of fusion. Because of these extensions, such lesions are often inadequately treated, and recurrences, therefore, are common. These epitheliomas are slow growing but when once disturbed by abortive or inadequate treatment are progressive and destructive. Meland stated that he employed protracted fractional radium therapy given at a distance over a fairly large area. He stated that success in treating recurrent carcinomas in these regions depends on the presence or absence of necrosis of cartilage and bone, which are secondarily infected. In cases in which adequate irradiation has been used but is not followed by healing, he stated, he prefers to treat the lesion by electrocoagulation. Meland reported 43 cases of epithelioma of the nasolabial fold and the results of treatment.

Simpson and Evans[7] described a case of pedunculated fibroma of the nose. The tumor was situated on the left side of the septum anteriorly and was excised along with the underlying cartilage. Simpson and Evans stated that granulomas, angiomas and fibromas are usually found in the anterior half of the septum, myxomas in the middle or posterior third, and chondromas in any part of the septum.

---

5. Furniss, F. W.: Mesial Dermoid Cysts of the Nose Occurring in Twin Boys, J. Laryng. & Otol. **53**:314-315 (May) 1938.

6. Meland, O. N.: Treatment of Epitheliomas of the Nasolabial Fold, Am. J. Roentgenol. **38**:730-739 (Nov.) 1937.

7. Simpson, W. L., and Evans, J. D.: Pedunculated Fibroma of Nasal Septum, Ann. Otol., Rhin. & Laryng. **46**:527-530 (June) 1937.

Hall,[8] in reporting 4 cases of true, or hard, papilloma of the nasal cavity, discussed the etiology, pathology and clinical aspects of the disease. Papilloma durum is histologically nonmalignant but shows great tendency to recur and may, if neglected, undergo malignant change. This occurred in 2 of Hall's cases. Papilloma durum almost always has its site in the anterior part of the nose, over the turbinates and septum, but may invade the sinuses. The only symptom is gradual obstruction of the air passage or a visible lesion. The most successful treatment is thorough destruction with surgical diathermy. There appears to be a tendency for the entire nasal mucosa to produce this growth, which may reappear in an entirely different region after it has been thoroughly eradicated. Irradiation has proved unsatisfactory. A good bibliography of this condition was appended.

Huizinga [9] reviewed 35 cases of malignant tumor of the paranasal sinuses observed in the Groningen University, with the object of outlining the frequent difficulties in making a correct diagnosis. Roentgenographically, definite erosion of the bone may be absent or difficult to determine, especially in the frontal sinuses, where the tumor in 5 of the cases was situated. Microscopically, it may be difficult to obtain tissue showing the actual growth, and only inflammatory tissue may be found. One case was described as an instance of carcinosarcoma, and Huizinga discussed the literature on this subject at length.

PHARYNX

Baker [10] presented a study of 72 carcinomas of the pharynx, including 6 in the nasopharynx, 39 in the oropharynx, and 27 in the laryngopharynx. He found that there were no pathognomonic symptoms and that the disease has an insidious onset. No attempt was made to evaluate the results of treatment, although Baker stated that the best results are dependent on early diagnosis.

In an excellent and detailed discussion on the relative value of radium and roentgen therapy in external irradiation for malignant lesions of the throat, Fulton [11] explained that radium is ideal from the standpoint of continuous irradiation but falls short in relation to physical distribution. Roentgen therapy, on the other hand, is ideal from the point of view of physical distribution but cannot be employed continuously. Since in

8. Hall, A.: Contribution à l'étude clinique du papillome dur des fosses nasales, Acta oto-laryng. **25**:226-239, 1937.

9. Huizinga, E.: Diagnostische Schwierigkeiten bei malignen Nasennebenhöhlentumoren, Acta oto-laryng. **25**:296-310, 1937.

10. Baker, D. C., Jr.: Carcinoma of the Pharynx, Laryngoscope **47**:904-908 (Dec.) 1937.

11. Fulton, J. S.: X-ray Therapy in Malignant Disease of the Throat, J. Laryng. & Otol. **52**:492-496 (July) 1937.

Fulton's opinion physical distribution is the more important factor, he expressed a preference for roentgen radiation. His patients with malignant lesions of the throat were given roentgen therapy for six weeks. During this time they were treated every day except Saturdays and Sundays for two hours in the morning and two hours in the afternoon. A detailed explanation of the subsequent reaction produced by such treatment was given.

Justras [12] reported the effects of treating 48 patients having pharyngeal lymphosarcomas with protracted roentgen therapy (long focal distance, well filtered rays, large bilateral fields, hourly weak dosage and daily exposure). The patients were treated at the Institute of Radium of Paris. He expressed the opinion that roentgen therapy is the most advisable for such cases. Among the 48 patients treated, he reported 80 per cent immediate local cures and 34.8 per cent survivals of five years and more. The extent of involvement of the lymphatic system is the most important clinical factor in determining the prognosis. Although the total dosage used caused radiodermatitis in some cases, the postradiation reaction did not go beyond this. Justras concluded that the complications due to roentgen rays properly applied are of little importance.

A lengthy, detailed and fine account of the treatment of cancers of the pharynx was given by Martin.[13] While he expressed a preference for fractional roentgen irradiation as developed by Coutard, he pointed out the value of combining such treatment with interstitial irradiation with radon in many cases. He stated that he employed radon seeds in the local lesion and in some cervical metastatic nodes. The use of the open mouth as a portal for additional treatment by roentgen therapy in many lesions within the mouth and the upper part of the pharynx was pointed out. In addition to an account of the technic employed, dose and methods of treatment for cancers of the nasopharynx, oropharynx and hypopharynx, Martin discussed the various complications of irradiation and procedures for preventing and treating such complications.

In a paper on malignant lesions of the nasopharynx, Furstenberg [14] stated that squamous cell carcinoma is the most common malignant neoplasm found in this region. Tumors of teratologic origin occasionally may be found in the nasopharynx. Early diagnosis depends on the recognition and proper interpretation of such signs and symptoms as

12. Justras, A.: Protracted Roentgen Therapy of Pharyngeal Lymphosarcoma, Am. J. Roentgenol. **39**:792-798 (May) 1938.

13. Martin, H. E.: Treatment of Pharyngeal Cancer, Arch. Otolaryng. **27**: 661-691 (June) 1938.

14. Furstenberg, A. C.: Malignant Neoplasms of Nasopharynx, Surg., Gynec. & Obst. **66**:400-404 (Feb.) 1938.

pain, involvement of the cervical glands and symptoms referable to the ear. Late manifestations of the disease include paralysis of the cranial nerves due to an invasion of the base of the skull by these lesions. Since the local lesion may be insignificant in size, may be hidden within the eustachian tube or may lie beneath the surface epithelium of the nasopharynx, the diagnosis may be difficult. Furstenberg had tried radical excision, electrocoagulation and roentgen and radium therapy in various combinations but declared that the prognosis remains hopeless. He stated that there appears to be no adequate means of arresting the upward invasion and consequent involvement of osseous tissue by these malignant tumors of the nasopharynx.

Needles [15] presented a study of 35 cases of nasopharyngeal malignant tumor, 16 of the patients manifesting neurologic complications. In pointing out the symptoms produced by such growths, he described a basilar syndrome due to involvement of the cranial nerves coursing through the middle cranial fossa. The symptoms referable to the cranial nerves may result also from meningioma, metastatic neoplasm, aneurysm or an inflammatory process, but they always call for an examination of the nasopharynx to rule out malignant tumor of the nasopharynx. Although the results of radiotherapy are encouraging, he said, in his opinion they are far from satisfactory. He stated that patients coming under his observation with a clinical picture of a nasopharyngeal malignant tumor are treated for the condition even though negative observations at biopsy are reported.

Gerlings and den Hoed [16] reported 47 cases of malignant tumor of the nasopharynx. The patients varied in age from less than 10 years to 80; 30 were men and the remainder women. The methods of examination, including inspection (anterior and posterior rhinoscopic examination), palpation, roentgenoscopic examination (to which great importance was attributed), biopsy and examination of the blood were carefully described. The various clinical types of this disease were enumerated, with their symptom complexes. The prognosis is unvaryingly poor. The treatment of choice was irradiation. Tables showing the type of tumor, frequency of symptoms and results of treatment were given. Eight patients remained free of symptoms from a year and a half to nineteen years.

---

15. Needles, W.: Malignant Tumors of the Nasopharynx, with Special Reference to the Neurological Complications: A Clinical Study of Thirty-Five Cases, J. Nerv. & Ment. Dis. **86**:373-398 (Oct.) 1937.

16. Gerlings, P. G., and den Hoed, D.: Diagnostic und Therapie der bösartigen Nasenrachengeschwülste, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:385-398, 1937.

Greifenstein [17] briefly reported a group of 31 reticuloendothelial sarcomas, the differentiation of which from lymphoepithelial carcinomas is sometimes difficult and to Greifenstein's point of view of little importance. Both types of tumor are found in the same locations, namely, the nasopharynx and the pharynx. The lymphoepitheliomas are characterized by a larger primary lesion and less tendency to generalized metastasis. Both are highly radiosensitive. The treatment of choice was believed to be Coutard's protracted fractional method.

Eigler and Koch [18] reported 8 additional reticuloendothelial sarcomas, of which 7 were in the tonsils and 1 in the maxillary sinus. In 1 case involvement was bilateral. Eigler and Koch disagreed with Greifenstein, who had expressed the opinion that these tumors do not tend to ulcerate and are slow growing. These lesions are frequently mistaken for inflammatory processes and subjected to surgical treatment, which is contraindicated. The treatment of choice remains irradiation. Of their 16 patients, 7 had died; 4 had remained well from a year and a half to six years, and the remainder were yet under observation. The difficulty of diagnosis was stressed.

Brunschwig and Tschetter [19] recorded the results of "periodicity" roentgen treatment in 14 cases of buccal and pharyngeal cancer. A periodic system of therapy is an economy of time, they said, entailing fewer days of treatment than a similar dose of fractionated roentgen therapy in daily consecutive treatments. With the periodic method, the systemic reactions were less, probably owing to the rest periods. However, Brunschwig and Tschetter stated that no evidence was obtained to indicate that the periodicity method as practiced might afford any marked difference in the results of treatment of far advanced and large buccopharyngeal carcinomas.

Levie [20] reviewed 147 cases in which malignant tumors of the tonsil were treated at the Clinic of Anton van Leeuwenhoekhuis in Amsterdam between the years 1916 and 1932. The cases were divided clinically into four groups which were found to correspond accurately with the histologic type of the growths. These groups were the following: (1) keratinizing carcinomas, (2) nonkeratinizing

17. Greifenstein, A.: Die Klinik der Retothelsarkome, dargestellt auf Grund von 31 eigenen Beobachtungen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **143**:189-215, 1937.

18. Eigler, G., and Koch, J.: Weitere Erfahrungen über das klinische und histologische Bild der Retothelsarkome, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**: 156-162, 1937.

19. Brunschwig, A., and Tschetter, D.: Experience with "Periodicity" Method of Roentgentherapy in Treatment of Bucco-Pharyngeal Carcinoma, Illinois M. J. **73**:54-56 (Jan.) 1938.

20. Levie, B.: Classification and Treatment of Malignant Tumors of the Tonsils, Acta oto-laryng. **25**:403-413, 1937.

carcinomas, (3) lymphoepithelial tumors and (4) sarcomas. The treatment consisted of surgical removal, with or without irradiation. The latter was found to give better results and is now exclusively employed. The Coutard method of treatment has not replaced the other technics but has not been in use long enough for an opinion to be formulated of its value. The results were as might be expected, better in the few cases in which no metastases were present. Thirteen patients survived five or more years. These results were compared with those of other clinics and found to be surpassed only by those of the Berven Clinic, which employs teleradiotherapy.

A case of extensive malignant involvement of the left side of the hypopharynx and the left cervical glands was presented by McNally.[21] Although the patient died eventually of pulmonary metastasis, a necropsy showed that Coutard roentgen therapy had caused the primary lesion and associated involvement of the cervical glands to disappear entirely. McNally pointed out that even though a patient with a carcinoma of the pharynx may have also an incurable growth elsewhere in the body, roentgen therapy is justifiable since it will relieve the patient of his distressing pharyngeal symptoms.

LARYNX

Jackson [22] described briefly the treatment of various benign tumors of the larynx. Condemning the practice of cauterizing the base of such benign tumors after their removal, he insisted that this type of treatment results in slight concavity of the vocal cord, which interferes with perfect phonation. He concluded that every benign laryngeal growth, even though symptomless, should be removed and stressed the importance of a microscopic examination in every case.

In a report of tumors of the larynx that can be removed under suspension laryngoscopy, LeJeune [23] limited his discussion to those laryngeal tumors of which he has taken colored motion pictures. He stated that in properly selected cases some very early intrinsic epitheliomas of the larynx can be treated under suspension laryngoscopy; the growth should be extirpated and its base thoroughly coagulated.

---

21. McNally, W. J.: The Effect of Deep X-Ray Therapy in a Case of Carcinoma of the Hypopharynx and Cervical Glands, J. Laryng. & Otol. **52**:555-559 (Aug.) 1937.

22. Jackson, C.: Treatment of Benign Tumors and Tumor-Like Conditions of Larynx, Tr. Am. Laryng. A. **59**:45-51, 1937.

23. LeJeune, F. E.: Tumors of the Larynx as Visualized by Suspension Laryngoscopy, Texas State J. Med. **33**:756-759 (March) 1938.

Klose [24] reported a case in which a congenital cyst of the larynx arising in the piriform fossa was removed by lateral pharyngotomy without an opening into the larynx. Also he described a case in which a lipoma arising from the vallecula and covering the upper aperture of the larynx was taken away by means of a snare.

Rey [25] reported a case in which an amyloid tumor of the larynx was removed by laryngofissure. One hundred and thirty-seven cases of amyloid tumor of the upper respiratory passages were collected from the literature. An extensive discussion of this rare disease and its etiology and pathology formed the main portion of the article. Letterer's complicated hypothesis as to the intervention of an antibody type of metabolic reaction in the presence of certain inflammatory or irritative stimulations in organs with a certain predisposition was adopted. The diagnosis may be assisted by vital staining with congo red. The condition is usually benign.

Foster [26] gave the results of treatment in 11 cases of laryngeal papillomatosis and his conclusions following the treatment. He expressed the belief that there is a distinct difference between the papillomas of children and those of adults. As to treatment, he recommended roentgen therapy of low voltage (100 kilovolts) to alternate sides of the neck at intervals from five days to a week. Good results were obtained by this method for children but not for adults. Foster expressed the opinion that if a large pedunculated mass of papillomas is present, especially in the anterior commissure, it is better removed. He stated that he performed tracheotomy only when there was sufficient dyspnea to make it advisable.

Although according to their statistics cancer of the larynx is curable in 82 per cent of cases, Jackson and Jackson [27] stated that gross statistics show that 90 per cent of patients with cancer of the larynx die of the disease. They reviewed 3 cases to prove this point. Their conclusions explained the discrepancy by the failure of most patients to consult a physician regarding their throats until the potentially curable stage of the disease is past.

A lengthy discussion of cancer of the larynx, especially of methods of treatment, was presented by Martin.[28] He divided cancer of the

24. Klose, W.: Zwei Fälle von seltneren Kehlkopftumoren, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:154-158, 1938.

25. Rey, W.: Zur Klinik der Amyloidtumoren der oberen Luftwege, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **143**:216-232, 1937.

26. Foster, J. H.: Treatment of Laryngeal Papillomatosis, Ann. Otol., Rhin. & Laryng. **46**:786-789 (Sept.) 1937.

27. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, S. Clin. North America **17**:1791-1795 (Dec.) 1937.

28. Martin, H. E.: Treatment of Cancer of the Larynx, Tr. Am. Acad. Ophth. **41**:213-268, 1936.

larynx into four varieties: (1) intrinsic, (2) extrinsic, (3) mixed intrinsic and extrinsic and (4) subglottic. In reference to treatment, he expressed the opinion that no single method, that is, surgical, radium or roentgen therapy, is suited to all clinical forms and histologic varieties of the disease. Attempts to cure early intrinsic carcinomas by implantation of radon through the laryngoscope were discouraged; such treatment, he said, should be reserved for advanced cancers which have extended widely, perforated cartilage or invaded the subcutaneous tissues. Martin stated that he never employed total laryngectomy, because of his belief that partial laryngectomy with radon implantation is as effective, with less morbidity and mortality. A detailed account of the following technical procedures was presented: partial laryngectomy, laryngoscopic treatment with implantation of a radon seed, endolaryngeal irradiation with the laryngostat, an instrument for holding a capsule of radium securely within the larynx after laryngoscopic manipulation, and the fractional or divided dose method of external irradiation and the implantation of radon seeds in metastatic nodes in the neck in extrinsic cancers. In closing he gave the results of treatment in his cases: Of 16 patients on whom partial laryngectomy had been performed, 3 died of postoperative pulmonary complications and 13 were alive from less than one year to six years after operation; of 32 patients on whom laryngostomy with implantation of a radon seed had been performed, 16 were alive and well from less than three years to more than ten years after operation, and of 31 patients who had suffered from extrinsic carcinomas, 4 were alive and well.

Galloway [29] expressed the opinion that irradiation is not the ideal treatment for every cancer of the larynx. He made an appeal for the early diagnosis of such lesions and recommended thyrotomy or partial laryngectomy in early low grade intrinsic cancers. For the more extensive malignant lesions, he expressed a preference for laryngectomy or possibly irradiation for some patients who find it scarcely possible to adjust their methods of living so as to live comfortably without a voice. He expressed the opinion that irradiation is indicated perhaps for intrinsic carcinomas of grades 3 and 4, on a basis of 1 to 4, and that it is practically always indicated for extrinsic laryngeal cancers. For postcricoid malignant lesions, he stated, he employed radiation as a therapeutic measure, but for localized malignant lesions of the epiglottis he used electrosurgical treatment under suspension laryngoscopy.

The surgical removal of cancer of the larynx is the most satisfactory, according to Looper.[30] He stressed the importance of early

29. Galloway, T. C.: Irradiation or Surgery in Cancer of the Larynx, Illinois M. J. **72**:276-279 (Sept.) 1937.

30. Looper, E. A.: Surgical Treatment of Cancer of the Larynx, South. M. J. **31**:367-374 (April) 1938.

diagnosis. For 20 per cent of early cancers, the conservative operation of laryngofissure can be employed. He reported cures of 80 to 85 per cent of such cancers. He stated that he used laryngectomy in cases in which laryngofissure cannot be employed and that he considered the former procedure as safe, with only a 1 or 2 per cent mortality. In performing a laryngectomy, he stated, he favored the use of rectal anesthesia and a crescentic cutaneous incision beginning above near the midline at the highest point of the right ala of the thyroid cartilage and curving downward and outward to the inner margin of the right sternocleidomastoid muscle and finally inward to the midline at the level of the first tracheal ring. He stated that he preferred a one stage laryngectomy unless the patient was badly dyspneic, in which event a preliminary tracheotomy was performed.

Schall [31] discussed laryngeal cancers which are too extensive for laryngofissure but still have not become extrinsic or metastasized to the cervical glands. He expressed the opinion that in such cases laryngectomy is the preferable method of treatment rather than irradiation. Four indications for laryngectomy were stated: (1) an intrinsic carcinoma, grade 1 or 2, of sufficient extension to make its operative cure by laryngofissure questionable; (2) involvement of the muscles or cartilages as evidenced by fixation of the cord or perforation of the cricothyroid membrane; (3) involvement of the posterior commissure, or (4) subglottic extension with involvement of the posterior commissure. Among his 25 laryngectomies, he had no operative mortality. Five of the growths were extrinsic and caused the death of the patients by recurring. Eighteen patients were alive without recurrences after periods varying up to six years.

In connection with the presentation of a moving picture, Palmer [32] gave a description of a case in which an intrinsic carcinoma of the right vocal cord was treated by laryngofissure.

Meyer zum Gottesberge [33] reported 26 cases in which laryngeal cancers involving the vocal cords were treated by laryngofissure. He advocated greater latitude in the use of this comparatively conservative operation. He discussed carcinoma involving the anterior commissure and the false cord, ventricle and subglottic region. Of these, the lesions involving the anterior commissure are the most favorable. The partial fixation of the involved cord need not necessarily exclude good results if the limitation of movement is found to be due to perichondritis rather than to direct involvement.

---

31. Schall, L. A.: Laryngectomy: Its Place in the Treatment of Laryngeal Cancer, Pennsylvania M. J. **41**:261-267 (Jan.) 1938.

32. Palmer, A.: Laryngofissure for Removal of Intrinsic Cancer of the Larynx, Laryngoscope **48**:202-203 (March) 1938.

33. Meyer zum Gottesberge, A.: Erweiterte Laryngofissur beim Stimmbandcarcinom, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:337-344, 1938.

Thomson [34] gave a complete account of subglottic laryngeal cancers and stressed the importance of early diagnosis. Since the typical subglottic carcinoma originates below or on the under surface of a vocal cord, it is poorly visible and produces few symptoms until it grows upward to involve the free edge of the cord. A few of the growths never involve the cord. Although some cause no fixation of the cord in many subglottic malignant lesions the amount of fixation is out of proportion to the tumor visible. When the growth first reaches the free edge of the vocal cord, it may produce a mouse nibble ulceration simulating a tuberculous lesion. There are no symptoms in the early stages. Slight dyspnea or stridor on exertion may be the first symptom, rather than hoarseness. The author compared the frequency and prognosis of these lesions with those of intrinsic or extrinsic laryngeal cancers. Treatment was given much consideration; laryngofissure or laryngectomy should be performed according to the situation and extent of the growth. For patients with large subglottic carcinomas whose age, health or prejudice forbids complete laryngectomy and for those for whom this procedure does not promise success, treatment by irradiation should be considered.

In reference to intrinsic laryngeal epitheliomas, whose location and character lend themselves to treatment by laryngofissure, Diggle [35] made an interesting comparative analysis of the treatment of such lesions by means of laryngofissure and by means of irradiation with the Finzi-Harmer technic. His report included 26 patients treated by himself and his colleagues between the years 1931 and 1935 inclusive. Of the 26 patients for whom the Finzi-Harmer method of irradiation was employed, 18 were under observation three or more years. These 18 patients were compared with 18 other patients treated by laryngofissure three or more years prior to 1933. The results of Diggle's study revealed that treatment by the Finzi-Harmer method of irradiation gives a lower operative mortality, with a greater risk, however, of recurrence or persistence of the disease as compared with treatment by laryngofissure. His statistical tables led him to state that it would appear that similar results can be achieved by either method of treatment, with or without fixation of the vocal cord and even with limited invasion of the anterior commissure. However, the original method of Finzi-Harmer irradiation seemed to be unsuitable for subglottic infiltrations. Finally, Diggle stated that he was convinced that the healing period is much shorter after laryngofissure, whereas the convalescence after irradiation is prolonged and tedious.

---

34. Thomson, St. C.: Subglottic Cancer of the Larynx, J. Laryng. & Otol. **52**:803-816 (Dec.) 1937.

35. Diggle, F. H.: The Treatment of Intrinsic Laryngeal Cancer, J. Laryng. & Otol. **52**:463-476 (July) 1937.

Simpson [36] described a new instrument, a modification of a Jackson bronchoscope, for the intralaryngeal application of radon. The instrument is inserted under local anesthesia and permits of direct inspection of the larynx so that a carrier holding the radon tube can be inserted directly into the larynx.

TRACHEA

Falconer [37] reported a case of amyloid tumor found accidentally at autopsy. The lesion was located at the bifurcation of the trachea and had given symptoms of obstruction before the patient's death. A brief and incomplete bibliography and a review of cases from the literature were given.

Moersch, Broders and Havens [38] described a case of tracheopathia osteoplastica (osteoma of the trachea), which is extremely rare. They stated that the majority of such growths produce no symptoms but a few may cause gradually increasing dyspnea, hoarseness, cough and expectoration. Usually occurring in multiple numbers, these tumors may involve any part of the trachea except the posterior membranous portion. The case reported involved a patient 40 years of age, who had complained of increasing dyspnea and hoarseness for fourteen years. Multiple hard nodules were found studding the wall of the trachea except for the posterior portion. Two of these were removed for biopsy. The lumen of the trachea was reduced to about a third of its normal size.

Four hundred and thirty-three primary tumors of the trachea on record in the literature to the end of June 1936 were reviewed by Culp.[39] One hundred and forty-seven, or 34 per cent, were carcinomas. To this series, Culp added 2 additional cases of primary carcinoma of the trachea. He found that the clinical symptoms of a primary tracheal carcinoma were about the same as those produced by any neoplasm of the trachea and that most of the patients died within one year of the initial examination. The highest incidence of carcinoma was in men between the ages of 40 and 60. The tumors were located most frequently in the lower third of the trachea on the posterolateral walls. Metastases were found in 68 per cent of the cases and were mostly regional. Adenocarcinomas and squamous cell carcinomas were of equal frequency; these two types were the most common. Culp stated that adenocarcinomas probably originate in the mucous glands of the trachea.

---

36. Simpson, F. E.: A New Instrument for the Intralaryngeal Application of Radon, Arch. Otolaryng. **27**:773-774 (June) 1938.

37. Falconer, B.: Ein Fall von Amyloidtumor der Trachea und der grossen Bronchien mit dyspnoischen Erscheinungen, Acta oto-laryng. **26**:353-357, 1938.

38. Moersch, H. J.; Broders, A. C., and Havens, F. Z.: Tracheopathia Osteoplastica (Osteoma of the Trachea), Arch. Otolaryng. **26**:291-293 (Sept.) 1937.

39. Culp, O. S.: Primary Carcinoma of the Trachea, J. Thoracic Surg. **7**: 471-487 (June) 1938.

Some of the squamous cell tumors were assumed to be new growths from embryonic rests of esophageal epithelium in the trachea, while others were assumed to be the result of metaplasia.

Keeney [40] gave a detailed report of a case of primary carcinoma of the trachea with cutaneous metastases in a man 73 years of age. He stated that cutaneous metastases are rare but occur more frequently with carcinoma of the trachea than with carcinoma of the lung or bronchi. The paper ended with a discussion of his explanation of parietal dissemination of carcinomatous implants.

Rathbone [41] described a case of primary carcinoma of the trachea. On a fluoroscopic examination of the chest and neck, the tumor was first discovered. It was removed through a bronchoscope, and the patient was then given roentgen therapy after the method of Coutard. The patient has been well for two and a half years.

Kleinfield and Smith [42] reported a case of lymphoepithelioma which involved the mediastinum; a portion of the growth projected into the posterior wall of the trachea above the bifurcation. Diagnosis was made by biopsies of tissue taken from the portion of the tumor in the trachea. Repeated mirror examinations of the nasopharynx for a primary focus gave negative results. However, by the use of a Yankauer tubular nasopharyngeal speculum several specimens for biopsy could be obtained from the nasopharynx, and these pieces of tissue were reported as lymphoepithelioma. The patient died shortly after these examinations had been completed.

ESOPHAGUS

Farrell [43] made a study of 100 cases of carcinoma of the esophagus to determine if a correlation of the clinical and roentgenologic findings might reveal signs by which carcinoma of the esophagus could be diagnosed earlier and at a time when it would be amenable to surgical treatment. However, no such correlation was discovered, because, as the author stated, patients come too late for examination; in 95 per cent of the cases the diagnosis of organic disease was made and the presence of a malignant lesion was suspected at the time of the first roentgenologic study. Some interesting facts concerning the locations in the esophagus, frequency and symptoms of such lesions were brought out.

---

40. Keeney, E. L.: Primary Carcinoma of the Trachea with Cutaneous Carcinomatosis, Bull. Johns Hopkins Hosp. **61**:411-420 (Dec.) 1937.

41. Rathbone, R. R.: Primary Carcinoma of the Trachea, Rev. Tumor Therapy **1**:66-68 (Aug.) 1937.

42. Kleinfield, L., and Smith, G. Y.: Lymphepithelioma of Mediastinum—Metastatic, Laryngoscope **48**:204-205 (March) 1938.

43. Farrell, J. T., Jr.: Integration of Clinical and Roentgenologic Findings in the Diagnosis of Carcinoma of the Esophagus: A Study of One Hundred Cases, Radiology **30**:412-416 (April) 1938.

A detailed account of the treatment of carcinoma of the esophagus was presented by Decker.[44] His conclusions revealed that neither surgical treatment nor radiotherapy offers benefits leading consistently to recovery. Better end results depend on early diagnosis and treatment. Selected patients suitable for surgical treatment should be offered this method of treatment even though the mortality is high. Radiotherapy should be chosen with curative intent for those who are unsuited for surgical treatment and as a method of palliation for persons who are beyond cure.

Moersch[45] reported a case in which an early stage of carcinoma of the esophagus was treated successfully by means of surgical diathermy. The patient has been perfectly well with no sign of recurrence for over two years. However, Moersch observed that for 447 patients with carcinoma of the esophagus whom he has examined, the average duration of symptoms before the patient came for observation was seven and one-tenth months. Thus, early diagnosis is difficult.

Garlock[46] reported 3 cases in which he treated carcinoma of the thoracic portion of the esophagus successfully by surgical resection. At the time that this paper was written, the first patient had been alive and well for eighteen months after operation; the second for eleven and a half months; and the third for two and a half weeks. The author stated that only 6 successful resections of the thoracic part of the esophagus had been reported in the literature previous to his article. He expressed the opinion that surgical intervention in these cases should be undertaken more often and said that the mortality is high because most of the patients are elderly and there may be associated organic disease of the heart, lungs, kidneys or arteries. Then, too, when the average patient with such a carcinoma of the esophagus comes to the surgeon he is in a bad state of malnutrition and dehydration, which adds to the surgical risk. Emphasis was placed on early diagnosis by a roentgenoscopic and esophagoscopic examination of every patient who complains of any deglutitive disturbance for two or three weeks. Careful preoperative care, consisting of a diet of high caloric content, one or two transfusions and a preliminary gastrostomy, was stressed. A detailed account of the surgical technic for resection of the thoracic portion of the esophagus was presented.

---

44. Decker, H. R.: Diagnosis and Treatment of Carcinoma of the Esophagus, Pennsylvania M. J. **40**:1038-1045 (Sept.) 1937.

45. Moersch, H. J.: Treatment of Carcinoma of the Esophagus by Means of Surgical Diathermy, J. Thoracic Surg. **7**:43-47 (Oct.) 1937.

46. Garlock, J. H.: Surgical Treatment of Carcinoma of the Thoracic Esophagus, with Report of Three Successful Cases, Surg., Gynec. & Obst. **66**: 534-548 (Feb.) 1938.

News and Comment

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The forty-fifth annual meeting of the society was held at the Drake Hotel in Chicago on June 9, 10 and 11, 1939.

Twenty-five candidates were elected to active fellowship. Dr. Chevalier Jackson was elected an emeritus fellow. Dr. Jackson has been a member of the society since 1901, was its president in 1911 and has always been one of its active and most distinguished members.

The following council members were elected for the ensuing year:

Class A—George M. Coates, Philadelphia
John J. Shea, Memphis, Tenn.
William Mithoefer, Cincinnati

Class B—Samuel J. Kopetzky, New York
H. Marshall Taylor, Jacksonville, Fla.
Millard F. Arbuckle, St. Louis

Class C—H. I. Lillie, Rochester, Minn.
James G. Dwyer, New York
Frank R. Spencer, Boulder, Colo.

The section meetings will be held in January 1940, as follows:

Eastern Section—Pittsburgh
Southern Section—Columbia, S. C.
Middle Section—Kansas City, Mo.
Western Section—Los Angeles

Subject to the approval of the council, the 1940 meeting of the society, under the presidency of Dr. Lee M. Hurd, will be held in New York early in June, and the 1941 meeting, under the presidency of Dr. J. Mackenzie Brown, will be held on the Pacific coast.

AMERICAN CONGRESS OF PHYSICAL THERAPY

The eighteenth annual scientific and clinical session of the American Congress of Physical Therapy will be held September 5 to 8 at the Hotel Pennsylvania in New York. Preceding this session the congress will conduct an intensive seminar in physical therapy for physicians and technicians on August 30 and 31 and September 1 and 2.

Registration is limited to one hundred and is by application only. Further information and a preliminary program may be procured by writing to the American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago.

Abstracts from Current Literature

Ear

TOTAL RECONSTRUCTION OF THE AURICLE. E. C. PADGETT, Surg., Gynec. & Obst. **67**:761 (Dec.) 1938.

Reconstruction of the auricle is notoriously difficult. Padgett has summarized the causes of failure, barring infection, as "(1) the inability to obtain structural support of the proper size and shape, (2) the failure to obtain adequate covering of the proper thickness, texture, and color, (3) difficulty in maintaining the new auricle at an upright level in comparison with the opposite ear, (4) the development of an improper divergent angle of the auricle from the head, and, finally, (5) a tendency for folding, shrinkage, and diminution in size."

Four cases of total reconstruction, 1 of which has been under observation for ten years, are presented. The method of correction consisted of three operative sittings with a total hospitalization of three weeks. The first hospitalization is about one week; the second, after two months, another week, and the last, after another month, a third week.

In the first operation, the rudimentary cartilaginous masses are removed and discarded. The mastoid bone above and posterior to the future meatus is hollowed out to imitate a cavum, and the hollow is lined with the remaining skin flaps. An appropriate piece of cartilage is then removed in the usual manner from the eighth or ninth rib and fashioned. After extensive undermining, the cartilage is inserted through a small incision posterior to and above the ear; the wound is closed, and a pressure bandage is applied. Along the side of the neck a flap is raised and tubed. Its lower end is split so that the wider piece will be adequate to cover the posterior part of the ear and the narrower one, about 12 cm. long and 1 cm. wide, will become the future helix.

Two months later an incision is outlined posterior to the buried cartilaginous mass, which is then turned forward. The posterior edge of the cartilage is trimmed. The wide tube is brought up, attached to the scalp above the ear and then folded out to cover the posterior surface of the cartilage. The end of the small tube is attached to the upper end of the ear and slowly unrolled at different periods to form a helix. The raw area overlying the temporal bone posterior to the ear is covered with a split graft overlying a modeling compound stent.

Five weeks later the lower parts of the tubes are severed, unused parts replaced and general adjustments made.

Padgett's experience with a case observed for ten years indicates making the reconstruction slightly larger than the opposite ear, as a slight amount of shrinkage does occur.

MILLER, Philadelphia.

OBSERVATION ON THE PATHOLOGY OF MÉNIÈRE'S SYNDROME. C. S. HALLPIKE and H. CAIRNS, Proc. Roy. Soc. Med. **31**:1317 (Sept.) 1938.

The pathologic changes in the temporal bones during Ménière's syndrome are described. Each of 2 patients died shortly after operation to section the eighth nerve. The pathologic changes in the temporal bones in the 2 cases may be summarized as follows: Gross dilatation of the endolymphatic system affected chiefly the scala media of the cochlea and the saccule. Degenerative changes were present in Corti's organ and in the stria vascularis in both cases; in the second case only scattered albuminoid coagula were present throughout the endolymphatic spaces. These tissue changes appear to represent serous labyrinthitis due to toxins reaching the labyrinth through the round window membrane and via the blood stream from some focus of infection. It is suggested that the symptoms of

labyrinthine disorder are due to a lowered functional state of the affected labyrinth caused by anoxemia of its end organs induced by a rise in the pressure of its endolymph.

CAMPBELL, Philadelphia.

PATHOLOGY OF CHOLESTEATOMA OF THE PARIETAL BONE. H. MELLER, Monatschr. f. Ohrenh. **72**:791 (Aug.) 1938.

The author describes a case of primary cholesteatoma of the middle ear and cholesteatoma of the parietal bone.

LEDERER, Chicago.

PATHOLOGY AND THERAPY OF ECZEMA OF THE EXTERNAL AUDITORY CANAL. H. MELLER, Monatschr. f. Ohrenh. **72**:959 (Oct.) 1938.

This review of an important practical subject brings no new facts to light.

LEDERER, Chicago.

COMPLAINTS AND DISTURBANCES IN THE ORGANS OF HEARING AND BALANCE OBSERVED AFTER INJURIES TO THE HEAD. A. ALEXANDER and R. SCHOLL, Monatschr. f. Ohrenh. **72**:1021 (Nov.) 1938.

The authors have examined 231 patients with concussion of the brain, 27 with contusion of the brain, 22 with fractures within the facial skeleton, 40 with fracture of the vault of the skull and 67 with fracture of the base of the skull. About 30 per cent of these patients had complaints referable to the cochlea or the labyrinth. Complaints about tinnitus and diminution of hearing were in proportion to the severity of the injury, as they were more frequent with severe than with slight injuries. In contrast, the complaints about dizziness were more or less independent of the severity of the injury, as they were to be found as frequently with severe as with slight injuries. Dizziness was more frequently complained of than difficulty in hearing, the relation being about 40 per cent to 60 per cent. The objective examination revealed a disease in the middle ear or in the cochlea in about 33 per cent of the cases and in the labyrinth in 30 per cent. As with the subjective symptoms, the incidence of objective disease of the cochlea increased with the severity of the injury. Therefore, in only 15 per cent of the cases of concussion of the brain did evidence of disease of the middle ear or the cochlea appear on examination, in contrast with 80 per cent of the cases of fracture of the base of the skull. In other words, in every seventh case of concussion of the brain with the complaint of tinnitus or diminution of hearing disease in the middle ear or in the cochlea could be demonstrated clinically, while among patients with fracture of the base of the skull who complained of tinnitus or diminution of hearing four fifths had disease in the middle ear or the cochlea. Among all patients with injuries to the skull 5.2 per cent evidenced disturbances within the cochlear apparatus even in clinical examination. These difficulties were most often produced by disease of the internal ear (in about 60 per cent of cases), which appeared either immediately after the injury or long afterward. Complete deafness after an injury was relatively rare. It was found in 0.26 per cent of the patients who had difficulty in hearing.

The incidence of objective diseases of the labyrinth was independent of the severity of the injury and fluctuated around 40 per cent. Among all patients with injuries of the skull, 6.7 per cent evidenced disturbances within the labyrinth even on clinical examination. Hyperirritability of the labyrinth was observed most often (in 5.15 per cent of the cases). As a rule it did not appear immediately after the injury, but a considerable time later. Loss of irritability was observed less often (in about 0.52 per cent of the cases). At the end of their interesting paper, the authors compare the percentage of the symptoms evidenced clinically from the cochlea and the labyrinth with the other symptoms in the cases. They emphasize that of all neurologic symptoms those indicating disease of the cochlea or of the labyrinth or of both organs were most frequently evidenced in these cases.

LEDERER, Chicago.

OPERATIVE INJURIES TO THE SIGMOID SINUS AND THEIR RESULTS. L. KLICPERA, Monatschr. f. Ohrenh. **72**:1059 (Nov.) 1938.

Operative injuries to the sigmoid sinus are not as dangerous as is usually believed, provided that all pathologic tissue within the mastoid has been thoroughly removed. Of 20 cases of surgical lesions of the sigmoid sinus recovery was uneventful in 17, although in 4 of these cases the lesion was made through a diseased wall of the sinus. Of these 17 cases the lesion was produced during the operation in 10 and at the end of the operation in 7. Therefore the time when the lesion is produced does not influence the prognosis. In 3 cases the accident was followed by sepsis. In all these instances the wall of the sinus was diseased. In these 3 cases also recovery occurred, after an operation on the sinus and the jugular vein. As far as the prognosis is concerned, it does not seem to make any difference in what manner the sinus is perforated, whether with the gage, with the sharp spoon, by a bony splinter, by a sequestrum or by a puncture of the sinus. Likewise the time of operating in cases of acute mastoiditis does not influence the prognosis. However, the prognosis is worse for older persons. Symptoms of passive hyperemia of the brain were found in 1 patient for whom an injured sinus was tamponed and in 1 for whom an incised sinus was tamponed.

When the sinus is injured the bleeding should be stopped by a tampon made as small as possible. The removal of that tampon is dependent on the clinical course. (When one reads this paper one gets the impression that an operative injury of the sinus is a harmless occurrence. This impression is erroneous, as no one can ever make a statement with perfect assurance that a lesion of the sinus will not be followed by sepsis. Surgical lesion of the wall of the sinus must always be considered a serious complication.)

LEDERER, Chicago.

BILATERAL OTOGENOUS THROMBOSIS OF THE TRANSVERSE, THE SIGMOID AND THE SUPERIOR SAGITTAL SINUS, THE INTERNAL JUGULAR VEIN AND THE INNOMINATE VEIN, THE SUPERIOR VENA CAVA AND THE AURICLE OF THE HEART. L. HOFFMAN, Monatschr. f. Ohrenh. **72**:1173 (Dec.) 1938.

A 1 year old girl became ill with high fever, bronchitis and acute otitis media (left side). Three weeks later a subperiosteal abscess over the left mastoid was incised, but the antrum was not opened. Nevertheless, the child improved and was dismissed from the hospital. But three weeks after the operation she returned in poor general condition, with high fever. These findings were considered to be due to the incomplete operation on the left mastoid, although there was evidence of slight otitis media on the right side. Thorough simple antrotomy was performed on the left side. The operation was of no avail; on the contrary, the child gradually became worse within the ensuing month. The veins beneath the scalp became dilated, indicating extensive thrombosis of the dural sinuses. The operative wound in the area of the left mastoid was reopened and the sinus exposed. There was no thrombus within the sinus of that side, and therefore the right mastoid process was opened; it revealed extensive sinus thrombosis. This operation failed to stop the progressive thrombosis of the sinuses, and one month after the last operation the child succumbed to a pulmonary embolus. Autopsy revealed the conditions mentioned in the title of the paper.

LEDERER, Chicago.

STREPTOCOCCIC MENINGITIS ORIGINATING IN THE MASTOID CURED BY ORAL AND INTRASPINAL INJECTIONS OF SULFANILAMIDE. HALPHEN, RENE MARTIN and BIGNOTTI, Ann. d'oto-laryng., November 1938, p. 1033.

In an 11 year old child streptococcic meningitis (organism in the spinal fluid) developed from recurring mastoiditis. Sulfanilamide was given, and the dose was based on the ratio of 0.15 Gm. per kilogram of body weight. It amounted to about 4 Gm. given by mouth at intervals of two or three hours and 15 cc. of 0.85 per cent solution of sulfanilamide injected into the subarachnoid space after prelim-

inary withdrawal of an equal quantity of cerebrospinal fluid. The concentration of sulfanilamide in the cerebrospinal fluid was kept at about 4 mg. per hundred cubic centimeters of spinal fluid. The concentration in the blood was kept at about the same figure.

The authors refer to about 7 cases in which streptococcic meningitis has recently been reported in the French literature as cured. They point out that the ideal treatment is surgical drainage and prompt and adequate sulfanilamide therapy with oral and subarachnoid administration. This should result in a cure in the large majority of cases. The dosage must be maintained for a sufficient length of time; else relpases tend to occur. The time may be from two to six weeks. This is best determined by the observations on the cerebrospinal fluid. Within four or five days organisms have generally disappeared from the spinal fluid. The concentration of sulfanilamide in the cerebrospinal fluid must be kept above 4 mg. per hundred cubic centimeters at the height of the disease. As a rule improvement in symptoms occurs within a few days. If the symptoms clear the dose may be carefully reduced.

The authors suggest that other treatment should consist of supporting measures, such as intake of fluid, careful check on blood cells and occasional transfusions.

McNALLY, Montreal, Canada.

Larynx

BRONCHO-ESOPHAGOLOGY: POSTULATED SEMASIOLOGIC OBSERVATIONS. CHEVALIER JACKSON, Ann. Otol., Rhin. & Laryng. **47**:1049 (Dec.) 1938.

In an address delivered at the meeting of the American Bronchoscopic Society in Atlantic City on April 30, 1938, the author makes a plea for the adoption of new terms which would more accurately define the work of the society, as well as indicate the larger aspects of the field of study.

The Greek suffix *-skopion* (from Greek *skopein*), modern Latin, *-scopium*, means "viewing"; it does not include the scientific knowledge acquired by viewing. In the twenty-one years that have elapsed since the society was founded, a mass of scientific knowledge has accumulated that merits the suffix *-logos*. This knowledge in part deals with the etiology, pathology, symptomatology, diagnosis, prophylaxis, prognosis and treatment of the tracheobronchial tree and of the esophagus.

The new terms referred to are bronchology and esophagology, and it is suggested that the name of the society be changed to American Broncho-Esophagologic Society. Many interesting points are brought forth in the article, with the following concluding remarks:

"In suggesting for consideration the changing of the name of this society it should be emphasized that it is not a matter of terminologic hairsplitting, nor is it a matter of semantics; but rather of (a) the establishment of a new department of study; (b) doing justice to our work; (c) conforming to the act of all other special medical societies in adopting a name indicating the field of work, or department of study, rather than the method or the instrument of examination."

GORDON, Philadelphia.

Nose

MALIGNANT NEOPLASMS OF THE NASOPHARYNX. I. JEROME HAUSER and DURWIN H. BROWNELL, J. A. M. A. **111**:2467 (Dec. 31) 1938.

The authors have reviewed in moderate detail the contributions of Gordon New and others to the study of malignant neoplasms of the nasopharynx and the varied symptoms that they can produce elsewhere. More careful study of the history, signs and other findings is apt to lead to more correct diagnoses of lesions of the nasopharynx, especially carcinoma, for the latter condition is said to be a relatively common disease.

Typical cases are cited, and a plea is made for thorough examination of the nasopharynx, including biopsy and histologic evaluation.

The authors close with the following conclusions:

"1. Malignant neoplasms of the nasopharynx often manifest themselves by symptoms which are extranasal in nature.

"2. Cervical swellings, changes in the tympanic membrane, unilateral deafness or stuffy sensation in one ear, pain in the head or throat, diplopia or rectus lateralis paralysis, or unilateral paralysis of any cranial nerves calls for careful examination of the nasopharynx.

"3. All malignant neoplasms arising from the lining epithelium of the nasopharynx appear to be medullary squamous cell carcinomas. Lympho-epithelioma and transitional cell carcinoma are believed to be highly undifferentiated forms of squamous cell carcinoma.

"4. Irradiation is at present the only form of treatment. In spite of poor results, if earlier diagnoses were made, chances of arresting or destroying the neoplasm would be greater.

"5. The nasopharynx should be given careful routine inspection by the oto-laryngologist."

GORDON, Philadelphia.

BIOLOGIC DETERGENCE BY THE PROETZ-LE MEE METHOD FOR OZENAL RHINOSINUSITIS. P. C. MONTI, Oto-rhino-laryng. internat. **22**:585 (Nov.) 1938.

In every case fetid atrophic rhinitis has been preceded by simple atrophic rhinitis. The biologic properties of mucous membranes depend on the constitution of the subject. It is known that vitamin C plays an important role in biologic and immunizing processes. After the fiftieth year of life the content of vitamin C in the organism is diminished. Schoenlank has shown an analogy between the pathologic changes in the mucosa in senescence and those provoked by essential hydropenia; he studied 200 cases of essential hydropenia and found a deficiency of vitamin C similar to that found in senescence. He thought that essential hydropenia is primarily a constitutional disorder and is accompanied by lack of vitamin C. It is evident that there can be no specific treatment of ozena. The general and constitutional factors should be corrected after excluding the metasyphilitic forms of the disease. Monti has used with satisfaction sulfurous inhalations, with instillations of vaccines in the air passages by the method of Proetz, with the apparatus of Le Mee. The antivirus modifies favorably the affected mucosa, and the prolonged bathing of the mucosa by the escape of the medicament from the sinuses maintains the humidity of the latter.

DENNIS, San Diego, Calif.

THERAPEUTIC EXPERIMENTS WITH IONTOPHORESIS FOR DISEASES OF THE NOSE. F. IPOLYI, Monatschr. f. Ohrenh. **72**:1127 (Dec.) 1938.

By histamine iontophoresis the crusts in atrophic rhinitis can be removed rapidly and gently. However, complete recovery cannot be attained. In cases of dry catarrh, the author states, the complaints are diminished, but a cure is not accomplished. In cases of neuralgic headache without involvement of the paranasal sinuses, iontophoresis produces good results. It is possible to desensitize patients with hay fever by histamine iontophoresis.

LEDERER, Chicago.

A CONTRIBUTION TO THE ETIOLOGY OF RECKLINGHAUSEN'S DISEASE WITH INVOLVEMENT OF THE MAXILLARY SINUSES. FRITZ URFER, Ann. d'oto-laryng., November 1938, p. 1047.

A case of granulomatous and cystic involvement of the left antrum in which a radical Caldwell-Luc operation was done is reported. The author does not consider a more extensive operation necessary. There was evidence of Recklinghausen's disease elsewhere in the body. An adenoma of the parathyroids was removed at

operation. The author points out that if palpation gives negative results an exploratory operation on the parathyroid region is justified. The antrum healed, and the calcium metabolism returned to normal.

McNALLY, Montreal, Canada.

Miscellaneous

REFRIGERATED CARTILAGE ISOGRAFTS. C. B. O'CONNOR and G. W. PIERCE, Surg., Gynec. & Obst. **67**:796 (Dec.) 1938.

The problem of securing a graft which is easily procured, can be kept indefinitely and is well tolerated by the recipient's tissues seems to be well solved according to the results of O'Connor and Pierce. Reporting an experience with 375 refrigerated isografts of cartilage, extending over a period of five years, the authors describe their method of keeping the grafts and make some interesting observations on characteristics displayed by the material after being introduced to repair defects in the contour of the nose.

The material is stripped of its perichondrium, washed thoroughly, placed in a sterile glass container and covered with a solution of 1 part of aqueous merthiolate (1:1,000) in 4 parts of sterile physiologic solution of sodium chloride. The authors recommend that an isograft should not be used until it has been refrigerated in the solution for at least one week and negative cultures of the solution have been obtained. For preparation of the cartilage, the solution was changed twice weekly for two weeks and then only once a week. The grafts were anywhere from a few days to two years old when used. It appears that this material is less likely to bend after insertion than is an autogenous graft. The authors had only 6 infections, and these were handled as autogenous cartilage is—i. e., by complete removal or excision of the exposed portion. The material can be interchanged regardless of age, sex, color, race or blood grouping.

MILLER, Philadelphia.

DISCUSSION OF THE VALUE OF PHYSICAL METHODS IN THE TREATMENT OF SUPPURATIVE CONDITIONS OF THE NOSE AND THROAT. A. M. ZAMORA and others, Proc. Roy. Soc. Med. **31**:883 (June) 1938.

A. M. Zamora discusses the value of various physical agents. He states that conductive sources of heat, such as foments and hot bottles or pads, act as counter-irritants and are not penetrating. The same applies to cold ultraviolet rays. He believes that the complexity of the air cells in the upper regions of the nose makes ionization unsuitable for treatment of sinal infections. The use of an air-cooled quartz mercury vapor arc lamp appears to be of value, and the variety of lamps and infra-red radiation units is suitable in the acute and subacute phases. Diathermy and the ultrashort wave electrical currents appear to have some value in selected conditions. The value of roentgen rays in sinusitis appears indefinite, and they probably are of no great benefit. It is believed that the helpful physical effects are limited to the changes in heat that are brought about in the tissues.

C. B. Heald believes that the claims for benefits by the use of short wave electrical treatments has been grossly exaggerated. He discusses in some detail the use of the various physical agents.

A. Eidinow states that there is now a means of securing deeper penetration of the visible rays by a process of sensitizing the tissue by means of dyes. He believes that it may become of value in the treatment of infections of the nose, throat and ear.

H. V. Forster goes into some detail in regard to the technical aspect of electrical therapeutic apparatus.

W. A. Troup emphasizes the value of infra-red rays.

P. Baumens further discusses the application of short wave therapy.

A. Friel says that zinc electrolysis is of value in making an opening through the nasoantral wall in cases of maxillary empyema. CAMPBELL, Philadelphia.

SYNDROME OF THE NASAL NERVE. C. DEJEAN and P. ARTIERES, Arch. Soc. d. sc. méd. et biol. de Montpellier **19**:9, 1938.

Symptoms of neuritis of the nasal nerve were observed in a woman aged 66, who complained of sudden paroxysms of ocular pain on the left side, which radiated to the zygoma, accompanied by transient desquamation of the corneal epithelium, marked lacrimation and profuse rhinnorhea. For the previous month one or more such attacks had occurred daily, lasting from two to three hours and regularly associated with redness of the external bulbar conjunctiva. Vasomotor phenomena secondary to irritation of the nasal ganglion are probably the basis of the symptoms. In this case the left nasal fossa was painted twice at an interval of a week with a mixture of equal parts of cocaine, menthol and phenol, after which the manifestations gradually lessened and disappeared. The therapeutic test is considered to have confirmed the diagnosis.

J. E. LEBENSOHN. [ARCH. OPHTH.]

DISEASES OF THE VESTIBULAR APPARATUS SEEN DURING THE EPIDEMIC OF GRIP IN THE WINTER OF 1937-1938 (PROBABLY A SIGN OF INCOMPLETELY DEVELOPED ENCEPHALITIS). E. ERDELYI, Monatschr. f. Ohrenh. **72**:1131 (Dec.) 1938.

The writer observed during the winter of 1937-1938, 11 patients who suddenly became ill with severe labyrinthine symptoms (dizziness and spontaneous nystagmus, particularly after movements of head), labyrinthine hyperirritability and disturbances in equilibrium. In 2 cases, tinnitus and diminution of hearing and of bone conduction were present. The prognosis in such cases is generally good. Erdelyi believes that the labyrinthine symptoms were produced by incompletely developed encephalitis in the area of the vestibular nuclei, as other neurologic symptoms were absent.

LEDERER, Chicago.

RHINOSALPINGOTYMPANIC CATARRH AND ITS TREATMENT BY INSUFFLOINHALATIONS OF THE THERMIONS OF SILVER AND COPPER. Y. DE KÉRANGAL, Oto-rhino-laryng. internat. **22**:529 (Oct.) 1938.

Kérangal reports the results of treatment with the ions of copper and silver in cases of tubotympanic catarrh. Sixteen observations on patients varying in age from 5 to 67 years are recorded.

He calls the treatment "aero-thermo-metallo-iono-insufflo-inhalations of the thermions of silver and copper." The apparatus of Atmik is used in conjunction with Itard's catheter and appropriate double nasal olives. No detailed description of the apparatus is given. The treatment comprises a tubotympanic stage and a respiratory stage. The tubotympanic phase precedes the respiratory if the eustachian tube is sufficiently patulous. The sessions last from three to ten minutes, the tubotympanic stage being one fourth as long as the respiratory phase. The thermions are insufflated into the middle ear through the catheter, under control with the auscultation tube. In the respiratory phase the patient inhales the thermions through the nose, the nostrils being completely stopped by the olives used on the conduction tube from the apparatus, and then, with the olives removed, the patient closes the nose with the fingers and insufflates the inspired air into the eustachian tube by the Valsalva method followed by that of Toynbee. The warm air, charged with thermions, activates the local submucous circulation, dries the exudates, excites leukocytosis and is germicidal.

This method has been used by Kérangal for the past ten years, and the results have been excellent in the majority of the patients. The hearing lost because of deficiency of the middle ear has been restored, but, naturally, not that lost because of disorder of the internal ear. Tinnitus, deafness and catarrh successively disappear.

DENNIS, San Diego, Calif.

CONTRIBUTION TO THE STUDY OF MALFORMATION OF THE FACE AND OF THE JAWS IN THE CRANIOFACIAL DYSOSTOSES. PIERRE DE GUNTEN, Ann. d'oto-laryng., November 1938, p. 1056.

Five cases are carefully described, with an analysis of the etiologic factors and a discussion of the literature. The article is well illustrated.

McNALLY, Montreal, Canada.

OCCURRENCE OF BOWEN'S DISEASE IN THE ESOPHAGUS. F. CAILLIAU and LOUIS LEROUX, Ann. d'oto-laryng., November 1938, p. 1076.

A precancerous condition of epithelium was described by Bowen in 1912. In other cases reported the site has been elsewhere in the body. The authors discuss precancerous conditions and the tissue changes involved in the transition from normal growth to overgrowth.

McNALLY, Montreal, Canada.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY*

THOMAS W. LEWIS, M.D., *President*

Regular Meeting, March 6, 1939

WALTER H. THEOBALD, M.D., *Secretary*

LARYNGOPTOSIS. Presented by DR. J. E. MULSOW, Milwaukee.

The term "laryngoptosis" means an abnormally low position of the larynx, which may be congenital, due to developmental deficiency, or secondary to pathologic changes. I believe that to these causes trauma should be added.

In the case reported, the patient had sustained an injury in an automobile accident several months prior to coming under observation. He stated that after the accident he had some difficulty in swallowing and noted a lump low down in the neck. Four or five months later he had coughing spells and difficulty in breathing and at times was hoarse. At no time did he expectorate blood. Physical examination gave essentially negative results. Roentgen examination showed the hyoid bone to be located at the level of the sixth cervical vertebra, about two vertebrae lower than the normal position. The thyroid cartilage was partly ossified and was situated at about the level of the seventh vertebra and tilted forward. At fluoroscopic examination during the process of swallowing the motion of the hyoid bone and the larynx appeared normal. No tumors were noted.

It was concluded that the condition was probably congenital, as no evidences of pathologic change were noted. Had it been due to injury, one would expect some disturbance in the motility of the neck, difficulty in protrusion of the tongue and a history of expectoration of blood after the accident.

DISCUSSION

DR. SAMUEL SALINGER: When I saw the title of Dr. Mulsow's paper I had a feeling that I had read somewhere in some journal about a similar case. I looked it up and found it, and now I want to tell about it because the question of trauma was brought up by Dr. Mulsow. The case to which I refer was reported by Dr. B. Langenbeck (Subcutaner Abriss des Kehlkopfes mit Luxation nach Unten, *Ztschr. f. Hals-, Nasen- u. Ohrenh.* **43**:350, 1938). A man was adjusting the bridle on one of a team of horses when the animal became unruly and lunged forward. The chain running from the bridle to the pole caught the man in the neck, forcing him against a tree. He was knocked unconscious, vomited and expectorated blood. For some time afterward he had difficulty in swallowing; he became emaciated and had a cough due to lack of control of the larynx in the act of swallowing. Roentgenograms revealed the thyroid cartilage separated from the hyoid bone and displaced downward about 3 fingerbreadths. Langenbeck stated that he was able to find only 1 similar case in the literature. In Dr. Mulsow's case, it seems to me, had the dislocation been due to trauma there should have been more typical symptoms, such as edema and hemorrhage, since the act of tearing the hyoid bone from the base of the tongue must necessarily involve much force. As for congenital dislocation, it is so rare that little is known of its causation. Therefore, when a case of this type is encountered, particularly if

*The scientific program was presented by members of the Milwaukee Oto-Ophthalmic Society, as guests.

there is a question of liability or compensation, it is important to know that at least 2 cases in which trauma was undoubtedly the cause have been reported and are a matter of record.

DR. PAUL HOLINGER: My associates and I have observed 2 cases recently in which it may be said that laryngoptosis was due to trauma. In the first case the patient had been lifted 16 feet (5 meters) off the ground by a belt which wrapped itself around his neck. In the second case the patient had been playing "Indian." In both cases the thyrohyoid ligament was torn, but in neither case was there a history of hemoptysis.

DR. J. E. MULSOW, Milwaukee: It seemed evident that my patient had no difficulty in swallowing. The movements of the head and the protrusion of the tongue were normal. There was no fibrosis; the tissues of the neck were smooth and soft. The anomaly appeared to be of congenital origin.

SPONTANEOUS HEMORRHAGE FROM THE EAR. Presented by DR. T. F. MCCORMICK, Milwaukee.

Spontaneous hemorrhage from the ear is apparently so infrequent that the standard texts make little or no reference to it. A review of the literature is followed by a report of the case of a child aged 2 years. A violent hemorrhage from the left ear occurred the day after incision of the right ear and twelve days after the onset of illness. About 500 cc. of blood was lost, and the child was hospitalized and given several blood transfusions. During the next few days the bleeding recurred at intervals of about twelve hours, stopping spontaneously on each occasion. It was felt that the bleeding was from the neighborhood of the jugular bulb, and three days after admission the mastoid was opened and cleaned out.

No further bleeding occurred, and except for minor incidents the postoperative course was satisfactory. The case is reported because any profuse spontaneous bleeding from the ear is unusual. The apparent source of bleeding is also unusual, and the procedure employed to control the hemorrhage is out of the ordinary.

DISCUSSION

DR. SHERMAN SHAPIRO: While I personally approve of Dr. McCormick's treatment, I wonder how bleeding from the inferior and the superior petrosal sinus, which would be below the site of the tamponade, was controlled in this case.

DR. HOWARD BALLENGER: Was any evidence of a dehiscence in the bulb found in this case?

DR. JOSEPH C. BECK: I wonder if Dr. McCormick has come across the report by Dr. M. A. Goldstein, of St. Louis, of a case which he called one of vicarious spontaneous bleeding, because there was spontaneous bleeding in the region of the jugular bulb during menstruation. The blue color could first be seen through the tympanic membrane. At first there was bleeding into the pharynx, with subsequent dissolving of the drum membrane and external bleeding that required packing. It was possible to stop the hemorrhage for a time, but once it did not stop, and the patient bled so that venous ligation was necessary, including blocking off of the lateral sinus.

I do not think that the condition is limited altogether to children. I think that the cases recalled from Politzer were cases of advanced tuberculous osteitis and that many cases of spontaneous hemorrhage from the ear are of the same type.

DR. SAMUEL SALINGER: I think that Dr. McCormick's procedure in his case was logical. He was mindful of the fact that profuse hemorrhage from the ear may be due to erosion of the carotid artery. In his case, however, there was no evidence of any infection in the throat from which an involvement of the carotid artery might have been expected. On the other hand, it is well known that erosion of a vein is extremely rare except as a result of fulminating virulent infections.

Nevertheless, in the presence of an aural infection and in the absence of any swelling of the throat he was forced to expose the sinus and pack it off. Since this procedure stopped the bleeding, it is safe to assume that the bleeding originated from the jugular bulb. However, it is impossible to regard this assumption as a fact without direct exposure and inspection of the bulb.

DR. T. F. MCCORMICK, Milwaukee: I think Dr. Salinger has summed up the subject rather well. In answer to Dr. Shapiro's question, it is true that the sinus was collapsed at a point above the entrance of the inferior petrosal sinus, but nevertheless the bleeding did not recur. Dr. Beck is right about Politzer's cases; the hemorrhages all occurred in suppurative processes of the ear of long standing. The 20 cases in which such hemorrhages occurred in children were reviewed by Dr. Pearlman and Dr. Salinger. As for whether there was any dehiscence in the bone, I do not know; I performed the usual mastoidectomy, exposed the sinus widely and packed it off.

RHINITIS CASEOSA. Presented by DR. O. P. SCHOOFS, Milwaukee.

Rhinitis or sinusitis caseosa is an uncommonly observed condition characterized by an accumulation within the nose or the paranasal sinuses of a grayish yellow to brownish, glistening, occasionally fetid, putty-like exudate. Microscopically the caseous masses are structureless, containing degenerated polymorphonuclears and fatty acid crystals. Bacteriologically, various organisms may be found, predominantly fusiform bacilli, spirochetes, small filamentous growths and various strains of staphylococci and streptococci. Three types are said to occur clinically. Widely diverse opinions are held as to the cause and mode of development.

The prognosis is excellent. Complete removal of the pultaceous débris and routine after-care result in a healthy nasal cavity, complete amelioration of symptoms and no tendency to recurrence.

Two cases are reported, in which complete recovery occurred. The treatment is described and the literature reviewed.

DISCUSSION

DR. ALFRED LEWY: All have observed cases in which a cheeselike discharge was associated with sinal trouble, and I do not think I should know how to distinguish a case of rhinitis caseosa. I should like to ask what the distinguishing characteristics are and what was shown by the microscope. I am sure that all observers have seen the discharge vary all the way from a watery to a thick and cheesy consistency, without diagnosing rhinitis caseosa.

DR. M. REESE GUTTMAN: Dr. Schoofs has, unfortunately, stolen my thunder. I was to have presented this subject before the society last November, but because of my absence in Europe the date of my presentation was changed to the forthcoming April meeting. Therefore, I believe that the secretary will have to withdraw my name from the program.

My associate and I have had experience with 3 patients. The first, seen by Dr. Beck in 1922, had bilateral polyposis. He was operated on and was not seen until twelve years later, when he was sent by his physician with a diagnosis of carcinoma of the antrum. He had a large brawny swelling of the left cheek, associated with fixation as well as protrusion and deviation of the eye. The wall of the left nasal chamber was distended to such an extent that it pushed the septum into contact with the opposite, or right, nasal wall. Roentgen examination disclosed a mass within the antrum. A tentative diagnosis of carcinoma was made, and he entered the hospital for a sublabial operation, the intention being to resect the growth and introduce radium. To our surprise we found instead of a tumor a large amount of foul, thick, cheesy débris. The antrum was cleansed and a large opening made into the inferior meatus. The patient made an uneventful recovery. Microscopic examination of the material showed no organic structure whatever. The bacteriologist reported a large number of streptococci and staphylococci.

The second patient had a history of repeated recurrences of polyps in the nose after surgical procedures. In his case also a radical sublabial operation disclosed the caseous condition.

The case of the third patient was interesting because of the short time in which caseous sinusitis developed. Just before I left we had occasion to see a woman with bilateral polyps associated with a nasal discharge and chronic bronchitis. Roentgen findings indicated extensive involvement of the antrums, as well as of the ethmoid sinuses. The patient was subjected to extensive resection of the polypoid membrane in the ethmoid sinuses and the antrum, the latter by sublabial incision. The findings at operation consisted of a thick, hyperplastic, polypoid mucosa associated with polyps. No evidence of any caseous material was noted at the time. The postoperative course was apparently uneventful. On my return four months later I found that the patient had been seen three months after operation with a swelling of the left side and a fistula into the skin of the cheek. Roentgen examination showed that the left antrum was blocked out. Reoperation sublabially disclosed the entire antrum filled with caseous, structureless, foul material.

Histologic examination in each of these 3 cases disclosed no organic structure, and bacteriologic examination revealed numerous streptococci and staphylococci.

Dr. Joseph C. Beck: What has become of the old term "pseudomembranous rhinitis with caseous formation"? The condition so designated was a common clinical entity in some of the European clinics, and it was not mentioned this evening.

Dr. Walter H. Theobald: I should like to inquire if any bacterial growth was found on culture of the discharge.

Dr. O. P. Schoofs, Milwaukee: The diagnosis is based on the discovery of a caseous secretion in the absence of tumor, tuberculosis or syphilis. The secretion is entirely homogeneous, occasionally containing degenerated polymorphonuclears. Greifenstein was able to produce the condition in rabbits by injection of various chemicals, principally turpentine. The lesion produced was not a pseudomembrane but consisted of intense subepithelial leukocytic infiltration, which did not necessarily involve the entire lining wall of the sinus. Greifenstein formulated his conclusions and said that further investigation must be made by the clinician. Wirth in discussing Greifenstein's report said that he was able to produce caseous rhinitis in rabbits and guinea pigs by inoculation with coccobacilli of the pseudotuberculosis type.

No cultures were taken in my cases. I believe that no definite conclusions can be drawn from the literature, although Greifenstein's work is rather convincing.

Injuries to the Sigmoid Sinus. Presented by Dr. E. Bach, Milwaukee.

Septic thrombophlebitis and its complications constitute the chief danger from injury to the sinus. Air embolism, while infrequent, has occurred. Possible injury to the lateral sinus during performance of a mastoidectomy is usually in the mind of the otologist, and extreme caution is observed. Precautions which might be observed include roentgen examination to disclose anatomic variations of the sinus the discovery of which would warrant modification of the operative technic. Extreme care is necessary in the use of the curet and the rongeur to avoid the production of splinters of bone, which may penetrate the sinus.

The accepted treatment for sinal injury is checking the hemorrhage immediately, exposing the sinus above and below the injury, and packing between the sinus and the bone with a gauze drain. The operation may then be completed and the mastoid packed in the usual way with the wound left open. If symptoms of sepsis supervene, ligation of the jugular vein, excision of the external wall of the sinus and removal of clots are indicated. Postoperative administration of sulfanilamide is of great advantage, as shown by the history in 1 of the 2 cases reported.

DISCUSSION

DR. ALFRED LEWY: In going over an experience of more years than I like to admit, I remember opening the lateral sinus or having it open, unintentionally, in 2 cases of perisinal abscess. In the first case the sinus opened when I was wiping off the granulations with gauze around my forefinger. In the second case the sinus opened while I was washing blood off my glove, and I do not know how it happened. The wall was evidently necrotic. I have seen it accidentally opened at operation when I was present but not operating, and I should say that the safest instruments are a sharp gouge and a mallet. I have seen sinuses opened secondarily after the use of a burr and have seen them opened accidentally by a curet and by a rongeur. The early stage of an operation, when the operator has come to the sinus without having a wide open space prepared first, constitutes a difficult problem. It is important to work in a widely exposed mastoid field so that one can see what one is doing. The curet must be used skilfully, and in my opinion the function of the rongeur is to remove the overhang and not the cellular tissue. If these rules are observed, I think, the sinus will rarely be accidentally opened.

DR. SHERMAN SHAPIRO: I want to commend Dr. Bach on his courage in reporting a mishap which hardly anybody fails to encounter at one time or another. I think the way one looks on this sort of accident depends on the consequences which it is one's fortune to meet. I know of 1 case in which an overenthusiastic resident tore the sinus during operation and fatal thrombosis developed in consequence of the accident. I would endorse everything Dr. Lewy said, although I do not believe that any instrument is fool proof or that this mishap may not occur to any one, under certain circumstances, because of a little lapse on the part of the operator or because of unforeseen conditions. At the Research and Educational Hospital my colleagues and I have taught residents, postgraduates and interns one thing which may minimize the danger, and that is to open the antrum and outline the trough between the posterior wall and the jugular vein first, before proceeding to the posterior part of the mastoid. In this way, if the sinus is accidentally opened, one at least knows that the major part of the operation has been accomplished and drainage established from the middle ear to the outside.

DR. ALFRED LEWY: I forgot to give all the details, and I should like to say that the method of procedure is the one advanced by Dr. Shapiro. I said that one should uncover the sinus first. I should have specified that the mallet and gouge should be used and that one must be careful. The edge of the gouge is smooth, with no nicks. A nick is capable of causing serious damage. After having uncovered the sinus and exposed the plate, the rest of the operation is done with the Lambard or the Kerrison rongeur.

DR. JOSEPH C. BECK: I suspected that Dr. Bach presented this paper with the thought that all those present should confess their sins, and I will confess. I have opened the lateral sinus accidentally twice, not in Chicago but both times in Cincinnati, once in the city hospital during a meeting of the Ohio State Medical Society and once in the late Dr. John Murphy's practice. One occasion was in a case of acute mastoiditis and the other in a case of chronic suppuration of the ear, in which in the course of radical mastoidectomy I entered the lateral sinus. In neither of these cases did anything serious happen. I am sure that many surgeons have seen a slight wound in the lateral sinus. Especially when streptococcic infection is not present and the patient is not very ill, one needs simply to protect the wound with petrolatum gauze, and nothing will happen. In both the cases I used the gouge, contrary to my usual procedure, which is to use the burr. I have always used the burr in mastoidectomies. I can see the sinus much easier and more clearly, and if I want to expose the sinus then I follow the suggestion Dr. Lewy made. I call the Lambard punch Beck's delight because it is the safest instrument I know of with which to get to the sinus. This is a learned paper, and every one should get marked benefit from hearing of the method Dr. Bach mentions for entering the sinus. One should not get panicky because it has

been accidentally opened, and it does not as a rule require immediate packing off above and below, because many small rents do not progress to sinus thrombosis but heal without further complications.

DR. L. BRACHMAN: This discussion recalls a case in which the condition had nothing to do with operation but was due to accident. My brother-in-law, about 30, suffered a fatal accident while dancing; he fell and hit the left side of the head, causing an extensive fracture, through the mastoid down to the base of the brain. He became cyanotic and within four minutes was dead. Postmortem examination revealed a fracture and extensive bleeding toward the base of the brain, which caused death. A rent into the sigmoid sinus caused the bleeding.

DR. FRANCIS LEDERER: I believe Dr. Bach is to be congratulated for presenting the error of omission which he recounted. One might say that it has been generally thought that one could expose the sinus with more or less abandon. That is the conception of Professor Neumann and, I believe, others. I believe it is rather a dangerous policy to assume that it does not make much difference whether one exposes the sinus or not. Dr. Bach did not give any further details of technic or state whether when he punctured the sinus he made a larger exposure. I believe it is rather good policy to expose the sinus further, perhaps having in mind that one can prevent complications and that—as he called to attention—other bony spicules may have become lodged between the bone and the dura of the sinus.

The point of dressings is interesting to me, especially when one exposes a perisinal abscess with granulations on the dura, which constitutes a weakened wall of the sinus. In such cases, especially in the treatment of contagious diseases, spontaneous or subsequent opening is not uncommon, whether because the gauze catches on the granulations and weakens the wall still further, it is difficult to say. It might be well to use petrolatum gauze in packing. Recently an article appeared (Klicpera, L.: Über operative Verletzungen des Sinus sigmoideus und ihre Folgen, *Monatschr. f. Ohrenh.* **72**:1059 [Nov.] 1938) in which the author called attention to a number of cases in which he exposed the lateral sinus and nothing happened, but I think it is bad policy to assume confidently that this will be universal. Dr. Beck rose in defense of the burr, but I assume that it is still a dangerous instrument around the lateral sinus, because there always remains a thin plate of bone beneath the burr and it is that last thin layer of bone that requires the most cautious treatment.

DR. J. C. SCHOOLMAN: I wish to say a word about the reliability of roentgen examination in regard to the position of the lateral sinus. An interesting case was observed about two weeks ago. Examination of the roentgenogram indicated ample room within the mastoid cavity, yet removal of the first piece of the cortex revealed what was apparently the sinus; on reexamination of the roentgenogram it was thought that it must be a pyogenic membrane. It was decided to treat it as a sinus, and much to the surprise of those in attendance it was a sinus, so forward and lateral that the usual operation had to be somewhat modified.

DR. E. BACH, Milwaukee: I think that Dr. Lewy and Dr. Shapiro in their discussions emphasized an important thing. I have seen mastoids with extensive coalescence and breaking down, and if one proceeds with a curet a deep cavity with steep overhanging walls may be produced. An accident to the sinus in a case like this would be a serious handicap. It would be hard to stop the hemorrhage and at the same time remove bone from the sinus above and below the injury. Dr. Lewy removes the mastoid cortex over a considerable area and then reaches the mastoid antrum by means of a funnel-shaped excavation. This is safer, gives better visibility and obviates difficulties in case of accident.

In regard to Dr. Lederer's question about what was done when the sinus was punctured, I wish to say that the bone chip measured about ¼ inch (0.6 cm.) in diameter. There was a small amount of bleeding. The chip was easily located and removed, but I did not make an extensive exposure of the sinus around the puncture. I was sure the chip was out, and when a small pack was put on the

sinus it stopped bleeding. Of course, it is known that when the dura or lateral sinus is exposed and a bone chip is pushed between the bony wall and left there postoperative complications are probable.

Dr. Schoolman's case is interesting, and such a structure is found occasionally. It is well to extend the incision forward when one encounters an anteriorly placed sinus, to expose the bony meatus and afford an anterior approach to the mastoid antrum.

CARCINOMA OF THE SPHENOID AND ETHMOID SINUSES. Presented by DR. T. L. TOLAN, Milwaukee.

Two cases of extensive carcinoma of the sinuses and adjacent tissues are presented, in which are exemplified (1) the necessity of a complete history and not merely an inquiry concerning the ear, nose and throat; (2) the advisability of a correlation of the history, the symptoms, and the physical laboratory and roentgen findings; (3) the desirability, when roentgen treatment is given for relief of pain in what appears to be a hopeless condition, of giving it in the same dose as if a cure were expected.

One of the cases reported is of interest because of the unusual site of metastasis.

DISCUSSION

DR. HOWARD BALLENGER: The first carcinoma reported on by Dr. Tolan probably was primary in the nose or the nasopharynx. However, it would seem to me that the second was metastatic in the nose, secondary to some other focus.

A few years ago I had occasion to look up the reported cases of primary carcinoma of the nasopharynx, and, if I remember correctly, regional metastasis, that is, metastasis to the cervical lymph nodes, occurred in most if not all of the cases. However, metastases to the viscera seldom or never occurred from carcinoma of the nasopharynx.

DR. FRANCIS LEDERER: It is always interesting to hear the history of a malignant tumor in a sinus, though one is seldom guided by the history, because the next tumor will run a different course. I should not feel bad about the subsequent course in the second case if I were Dr. Tolan, because it would have been difficult to anticipate that a rectal carcinoma would metastasize where it did, though one might readily understand that one from the liver or lung could. This type of case is always of interest, because, to my mind, the nasopharynx is one of the fields of investigation neglected in the course of routine examination. That is exemplified on numerous occasions. Posterior rhinoscopic examination augmented by use of the Yankauer speculum is particularly valuable in visualizing this space for early diagnosis. I have in mind a patient who last July complained of pain in the neck and in the left ear. A cloudy antrum was found and thought to be the basis of his trouble. Little attention was paid to the fact that he complained of pain in the neck. There was ptosis of the eyelid on the right side. Later on it became bilateral. Various diagnoses were not proved. The patient had been in the clinic for some time, and not much attention was paid to the nasopharynx. One day, recalling that Dr. Furstenberg in reporting a number of cases had stated that the nasopharynx was a neglected area, I carefully examined the nose and nasopharynx. I found a mass which proved to be carcinoma. The specimen as observed at autopsy was interesting, for it was possible to trace the origin of the tumor and its invasion of practically the same area that Dr. Tolan described as invaded in his second case. The case will be reported in detail at a future time. I have appreciated hearing this report on carcinoma, but I cannot feel that taking a specimen for biopsy stirred up metastases. It would not have made much difference, as it appears now, but I think it is dangerous even to admit the possibility, because it is sufficiently difficult to convince laymen that biopsy should be done—to say nothing of physicians. Therefore, I should certainly disregard that possibility and proceed in the next instance to try to obtain a specimen for biopsy.

DR. JOSEPH C. BECK: I want to compliment Dr. Lederer on his discussion, particularly the attitude he takes toward biopsy. This principle of diagnosis has always been my thought in teaching, and I am glad he brought it out.

DR. T. L. TOLAN, Milwaukee: It was interesting to find in discussing with the group of general surgeons the case of metastasis from carcinoma of the rectum that no similar case had been observed or heard of. I have not thought that taking the specimen for biopsy stirred up metastasis in the liver and the lung, as the symptoms and signs were evident too soon after surgical intervention.

TRANSVERSE FRACTURE OF THE PETROUS PYRAMID. Presented by DR. W. E. GROVE, Milwaukee.

Fractures which involve the petrous pyramid are classified as longitudinal or transverse, the former being more common. The former never involve the labyrinthine capsule directly; the latter always damage the capsule. Transverse fractures vary considerably in position and direction. The internal fracture courses through the internal auditory meatus and shatters the cochlea in its anterointernal portion, while the external passes through the entire internal ear, cochlea, vestibule and fallopian canal. When the middle ear is injured the damage affects only its medial wall. Fissures between the windows are frequently found; the promontory may be torn loose; the footplate of the stapes may be luxated.

When bleeding from the ear is associated with total loss of function, the fracture is both transverse and longitudinal. Usually it causes complete loss of cochlear and vestibular function, although retention of one or the other function has been demonstrated. Roentgenographic demonstration of the longitudinal fracture is difficult even when attempted early and after a month or two becomes practically impossible. The transverse fracture can usually be seen by roentgen examination even when microscopic, and this is true for many years after injury, because the capsular fracture does not close by bony union or does so imperfectly. When the line of fracture puts the internal ear into communication with the middle ear, the danger of late meningitis is always present. Cases have been reported in which such meningitis occurred as late as sixteen years after the fracture.

In the case reported, a transverse fracture of the left petrous pyramid resulted in total cochleovestibular paralysis on the left side and almost total loss of cochleovestibular function on the right side. The latter was probably due to massive hemorrhage into the right labyrinth, although injury to the nerves at the internal auditory meatus could not be excluded. The petrous fracture was demonstrable by roentgen examination eight and one-half years after it occurred.

DISCUSSION

DR. ALFRED LEWY: I should like to ask if there was any danger of paralysis of the facial nerve.

I should like also to say that such fractures are usually the result of automobile accidents and later lead to difficult legal complications.

DR. PAUL CAMPBELL: The only thing I should like to bring out is the difficulty of trying to diagnose these fractures by roentgen examination. I have made a study of all fractures of this type that I have had access to, and the astonishing thing is that many do not show in the roentgenogram even though every care is taken. I recently saw a fracture of the petrous pyramid with gross evidence of a fracture line going to the outside. Yet it did not show in the roentgenogram. Autopsy was performed after meningitis had developed several months later. There was a fracture in that very place, although it had not shown in the roentgenogram. I should like to ask what percentage of fractures Dr. Grove feels can be shown by roentgen examination.

DR. W. E. GROVE, Milwaukee: Dr. Lewy spoke of paralysis of the facial nerve with this type of injury. It did not occur in any of the cases which I reported. However, from a study of a mass of material and of the literature,

I can say that approximately 50 per cent of labyrinthine fractures are complicated by paralysis of the facial nerve, which is more likely to remain permanent than when it occurs after longitudinal fractures, although even after transverse fractures recovery from the paralysis occurs in a large percentage of cases.

Dr. Campbell spoke of the difficulty of getting satisfactory roentgenograms in such cases. I should say that basal fractures of the longitudinal type, that is, fractures of the middle fossa running along the anterior border of the pyramid, are much more difficult to show roentgenographically than are those of the transverse type. If the roentgenologist wil ltake pains to view the patient in a number of different positions, such a fracture can be shown. Stenvers' position is the best to use for the determination of labyrinthine fractures, because it exposes the pyramid excellently. Sometimes taking a picture at a slightly different angle will bring out the fracture; sometimes when its presence is suspected it cannot be shown roentgenographically. However, as there is another type of injury producing the clinical signs of which I have spoken, I do not know that one can definitely say the fracture is there simply because cochleovestibular paralysis occurs unless one can perform an autopsy. Ulrich presented a useful study of the question a number of years ago and showed that injury to the eighth nerve before entrance into the internal auditory meatus can be so severe as to produce the same clinical picture that fracture can produce. So if one cannot show the fracture in the roentgenogram one cannot say whether it is there or not, but I should say offhand that probably 50 per cent of the transverse fractures can be shown roentgenographically, although for the longitudinal type the percentage is much smaller.

Book Reviews

Test Words for Deaf People. By D. B. Fry, B.A., Lecturer in Phonetics, University College, London, England, and P. M. T. Kerridge, Ph.D., M.R.C.P., Lecturer in Physiology, University College, London, England. Price, 4s. 3d. London: H. K. Lewis and Co., 1939.

The authors have published in connection with their paper in the *Lancet* (Tests for the Hearing of Speech by Deaf People, *Lancet* **1**:106 [Jan. 14] 1939) a series of six large cards containing lists of words and sentences to be used in testing the hearing of those deafened to speech.

Considerable early work has been done on the phonetics of speech, which apparently has not greatly influenced the authors in selecting their list of words. Although the words used are all monosyllables, many of them seem to be difficult acoustically and are not included in the lists of the most commonly used (thus the most familiar) words. The latter item seems most important in the testing of children—the test words must be in their vocabulary. It would have been valuable, it seems, had the authors noted, at least on a key chart, the acoustic (frequency) values of the words used, as is done in Dayton C. Miller's early work (Science of Musical Sounds, ed. 2, New York, The Macmillan Company, 1922) and in Fletcher and Steinberg's classic paper on speech hearing (Articulation Testing Methods, J. Bell Laboratories). Other workers in this field, Wolff (cited by Andrews, B. R.: Auditory Tests, *Am. J. Physiol.* **15**:14, 1904; **16**:302, 1905. Andrews attempted to apply Wolff's work to the English language) and Marage (L'audition et ses variation, Paris, Beaugency), have stressed the importance of the acoustic study of the test words used.

The sentence lists, being simple and familiar, are serviceable, for example in testing the efficiency of an aid used by a deafened person. Five cards are used for sentences, and with them one should obtain a fair rating of the patient's mental agility, speed of interpretation and accuracy. It must, however, be noted that in all this testing of intelligibility and articulation the factor of loudness enters to such an extent that unless levels of intensity are maintained as by recorded speech the findings are vitiated by the inability of the tester to deliver speech on an even level.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.
Place: Brussels, Belgium. Time: September 1939.

Hungarian Otolaryngological Society

President: Dr. V. Zimányi, Zárda-u. 48, Budapest II.
Secretary: Dr. G. Kelemen, Reáltanoda-u. Budapest IV.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

Société Française d'Oto-Rhino-Laryngologie

Secretary: Dr. Henri Flurin, 19 Avenue Mac-Mahon, Paris, 17e.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. A. W. Proetz, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: New York. Time: June 10-14, 1940.

American Academy of Ophthalmology and Otolaryngology

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Bronchoscopic Society

President: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Secretary: Dr. Paul H. Holinger, 1150 N. State St., Chicago.

American Laryngological Association

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lee M. Hurd, 39 E. 50th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: New York. Time: June 1940.

SECTIONS:

Eastern.—Chairman: Dr. John R. Simpson, Medical Arts Bldg., Pittsburgh.
Place: Pittsburgh. Time: January 1940.
Southern.—Chairman: Dr. Walter J. Bristow, Doctors Bldg., Columbia, S. C.
Place: Columbia, S. C.: Time: January 1940.
Middle.—Chairman: Dr. Sam E. Roberts, Professional Bldg., Kansas City, Mo.
Place: Kansas City, Mo. Time: January 1940.
Western.—Chairman: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles. Time: January 1940.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Horace Newhart, 527 Medical Arts Bldg., Minneapolis.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.